**Open Access Library Journal**
2025, Volume 12, e14055
ISSN Online: 2333-9721
ISSN Print: 2333-9705

# Repercussions of Osteoporosis in the Oral Cavity

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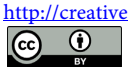
**How to cite this paper:** Ziyati, O., Sidqui, A., Hermas, S. and Sidqui, M. (2025) Repercussions of Osteoporosis in the Oral Cavity. *Open Access Library Journal*, **12**: e14055.
https://doi.org/10.4236/oalib.1114055

**Received:** July 31, 2025
**Accepted:** November 25, 2025
**Published:** November 28, 2025







## 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 increased risk of fracture, whose incidence increases with the age. Approximately 40% of women aged 50 years or older are at risk of 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 the menopause are the two principal determinants of the osseous loss. The factors of risk are mainly the family antecedents and the oestrogenic deficiency. The purpose of the preventive treatments of the 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 fabrics of support 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. It is a particularly worrying condition, as its frequency has increased in recent decades due to the aging population.

Ensrud *et al.* demonstrated [1] that there is a statistically significant difference between the mean number of missing teeth in osteoporotic patients [2] [3] compared to normal patients [4] [5]. They also reported that osteoporotic patients have lower bone mass and density, as well as a thinner cortex at the gonion. They also demonstrated a significant relationship between osteoporosis and alveolar ridge resorption. 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.

The alveolar bone is the bone most early and most severely affected by the process of physiological osteopenia. The latter is characterized by a decrease in the thickness of the alveolar ligament wall, the appearance of irregularities on the alveolar surface, a rarefaction of the bone trabeculae with the appearance of porosities, and a decrease in the quantity of osteogenic cells, a sign of decreased bone turnover.

## 3. Impact of Osteoporosis in the Oral Cavity

The systemic nature of osteoporosis results in distinct and measurable manifestations within the orofacial region.

### 3.1. Maxillary Bases

Among the oral structures, the maxillary bones are a primary site for osteoporotic changes, which are detailed in this section.

## Topographical Areas

The impact is evident in specific topographical areas in the maxilla and the mandible, as detailed in the subsequent findings.

**1) Maxilla**

It has been shown [6] that, in healthy women, there is a significant correlation between bone mineral density at the alveolar process of the maxilla and that of the alveolar process of the mandible of the lumbar spine, the coxae, and the radius, and that this bone mineral density at the maxillary level decreases with age [1] [6].

Ensrud *et al.* [1] also showed that the anterior maxilla represents the most sensitive site for distinguishing osteoporotic patients from normal patients due to the presence of significant amounts of trabecular bone and the relatively low cortical thickness in this region.

**2) Mandible**

**Mandible Angle: Gonion:**

Ensrud *et al.* [1] suggested that a 1 mm decrease in gonion height is an indicator of reduced bone mineral density.

Ensrud *et al.* [1] found a correlation between cortical thickness at the gonion and bone mineral density at the ulnar and spinal levels.

Other studies, Ensrud *et al.* [1], have shown that there is a significant difference in cortical thickness at the gonion between osteoporotic women and normal women of the same age.

Ensrud *et al.* [1] reported that osteoporotic patients have lower bone mass and density as well as a thinner cortex at the gonion. Lower mandibular border:

Numerous studies [6] using intraoral X-rays in osteoporotic patients to assess the mass and morphology of the maxillary bone bases have shown that the mandibular body is probably the site most affected by osteoporosis. Studies [6] have demonstrated that panoramic X-rays represent a very useful tool in the diagnosis of osteoporosis.

Indeed, in these patients, a change in shape and a decrease in the width of the lower mandibular cortex is noted; osteoporotic subjects present greater erosion at the lower mandibular border than in normal subjects (**Figure 1**).

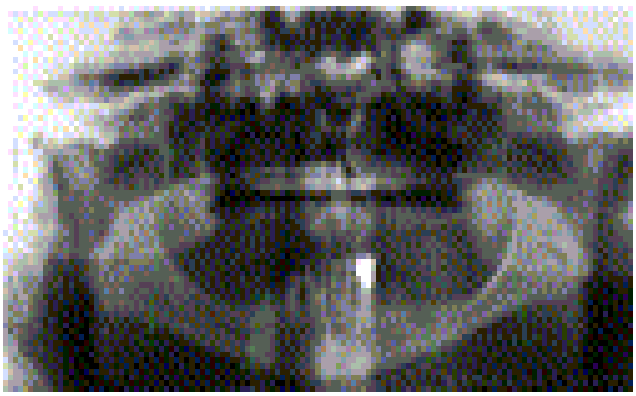


**Figure 1.** Panoramic radiograph showing the sites most affected by bone resorption in an osteoporotic patient (M. SIDQUI).

**Mandibular Condyles:**

In temporomandibular dysfunction, tissue changes and bone resorption occur in both the condyle and temporal components of the temporomandibular joint and can range from a simple decrease in cortical bone mass to severe destruction of the condyle and temporal components.

Ensrud *et al.* [1] reported that habits and conditions caused by decreased skeletal bone mass may cause functional disharmony of the masticatory system and increase the risk of temporomandibular disorder. They reported [1] the impact of osteoporosis in two subjects with temporomandibular dysfunction and suggested that early detection and antiresorptive therapy for osteoporosis deserve future consideration regarding the treatment of temporomandibular dysfunction. It has also been stated that osteoporosis may be the most significant risk factor for failure of initial surgery for pain and temporomandibular dysfunction [1] [7].

### 3.2. Alveolar Bone

Regarding alveolar bone loss, a study by Bandrivsky *et al.* [8] showed that 30% of male and female patients aged 65 to 69 are completely edentulous. The authors hypothesized that some of the lost teeth are due to excessive alveolar bone resorption, which may reflect bone loss due to osteoporosis. Alveolar bone loss in the jaws parallels the loss of initial bone mass. Although not directly related to osteoporosis, alveolar bone loss occurs following dental extractions. These procedures reduce both the remaining bone mass and the long-term chewing prognosis.

Two concepts should therefore be remembered:

- A certain proportion of lost teeth is due to excessive osteoporotic resorption of the dental alveolar support.
- Any dental extraction reduces the remaining alveolar mass.

Numerous studies [6] have demonstrated a relationship between alveolar bone resorption, the number of remaining teeth, periodontal status, and osteoporosis, through the measurement of bone mineral density at the skeletal and alveolar levels.

Ensrud *et al.* [1] suggested that a decrease in cortical thickness of 1 mm at the gonion is an indicator of a decrease in skeletal bone mass. It has been shown [1] that there is a significant correlation between cortical thickness at the gonion and bone mineral density at the ulnar and spinal levels. The same study showed a significant difference in mandibular cortical thickness between normal and osteoporotic subjects.

Ensrud *et al.* [1] also reported that osteoporotic patients have lower bone mass and density, as well as a thinner cortex at the gonion.

Bone is one of the primary targets of estrogen deficiency. In postmenopausal osteopenia, bone formation is decreased by a reduction in the number of osteoblasts, and resorption is increased by an increase in the number and activity of osteoclasts. Estrogens inhibit bone resorption and are thought to act directly on bone cells (estrogen receptors have been discovered on osteocytes, osteoblasts, and

osteoclasts).

Since bone turnover is greater in alveolar bone than in long bones, a systemic imbalance between apposition and resorption is thought to strongly affect it.

A study by Bandrivsky *et al.* [9] showed that:

In the groups (osteoporosis/osteopenia versus normal bone mineral density), women with estrogen deficiency exhibited reduced crestal bone density.

In the osteopenia/osteoporosis group, estrogen deficiency was associated with an almost doubled incidence of crestal bone density loss compared to women receiving hormone supplementation. But even though it seems relevant that estrogen deficiency and osteoporosis/osteopenia may have repercussions at the alveolar bone level, there is currently no evidence to support a link between reduced alveolar bone density and the progression of periodontitis in some patients.

### 3.3. Tooth Loss

Several studies have reported a significant correlation between osteoporosis and tooth loss (Table 1).

Table 1. Relationship between tooth loss and bone mineral density [14].

| Authors | Population | Results | Study Type |
|---|---|---|---|
| **Krall *et al.* (1996)** | 189 postmenopausal women without hormone therapy. | Correlation between decreased bone mineral density and the risk of tooth loss. | 5-year longitudinal study controlling for smoking, bone mass index, number of teeth, and treatment since menopause. |
| **May *et al.* (1995)** | 608 men, 874 women. (65 to 76 years) | - In men, the number of teeth is associated with bone mineral density but is independent of age, weight, and smoking.<br>- In women, no correlation. | Crossover study. |
| **Taguchi *et al.* (1995)** | 269 men and women. (83 - 88 years) | The number of teeth is greater in women over 60 years old with greater cortical thickness at the chin level. | Crossover study. |
| **Taguchi *et al.* (1995)** | 64 women. (50 - 70) years) | Radiographic evidence of lumbar fractures in women with a low number of teeth. | Crossover study. |
| **Krall *et al.* (1994)** | 329 postmenopausal women. | Correlation between the number of teeth and bone mineral density. | Crossover study. |
| **Kalemetti *et al.* (1994)** | 227 postmenopausal women. | No correlation between the number of teeth or bone height and systemic bone mineral density. | Crossover study. |
| **Elders *et al.* (1992)** | 286 women. (46 - 55 years) | No relationship between the number of missing teeth and bone mineral density. | Crossover study. |
| **Daniell (1983)** | 218 women. (60 - 69 years old) | Denture loss was three times more common in women with extreme metacarpal thinning. | Cross-sectional study. |

A prospective study by Herbert *et al.* [10] showed that it was possible to prevent the consequences of osteoporosis on the skeleton (femoral neck fracture, vertebral compression fractures, and wrist fractures) through calcium and vitamin D supplementation alone.

Unfortunately, no prospective study had investigated whether the same supplementation would have the same preventive effects.

Researchers at Tuf University Boston, Herbert *et al.* [10] took 145 subjects over the age of 65, divided them into two groups: the first supplemented with 500 mg of calcium and 700 IU of cholecalciferol-vitamin D3, the second on a placebo, and followed them at regular intervals during the three-year study period, and for another two years afterward. Of course, to avoid any bias, the researchers measured dietary calcium intake every six months.

Of course, the results were weighted to account for possible confounding factors: age, gender, smoking, education level, and diabetes. All these precautions are unnecessary when trying to confirm a cause-and-effect relationship. The results are spectacular: a 60% reduction in tooth loss among subjects supplemented during the intervention study. As always, this study should be confirmed by others before definitively asserting the role of calcium supplementation on tooth loss in old age.

As for the preventive effect of estrogens prescribed at menopause (MHT) on tooth loss, it remains purely hypothetical today. In the latest issue of the Archives of Internal Medicine (Balshi *et al.* [11], researchers from Washington University in Saint Louis (USA) reported that they followed 135 women, half of whom received calcium and vitamin D and the other half received estrogen therapy combined with vitamin-calcium supplementation, using retroalveolar or panoramic images. In the first group, alveolar bone density increased by 1%; if estrogen was added, the increase reached 1.8% [10].

Recent studies [6] have established the relationship between bone density and tooth loss and have shown that women with lower bone density tend to have fewer teeth.

### 3.4. Bone Healing

Histomorphometric studies [12] have shown that remodeling is normal in most osteoporotic patients, which may reflect a cyclical fluctuation of the disease; thus, at the time of diagnosis, bone metabolism may have returned to normal.

Observations of osteoporotic bone fractures have shown that they heal normally, suggesting that the repair processes in osteoporotic patients remain satisfactory [12].

The orthopedic literature [13] has reported that fractures in osteoporotic patients heal rapidly, and that bone mass levels and parameters associated with bone remodeling are largely consistent between osteoporotic patients and normal individuals.

Observations of osteoporotic bone fractures have shown that they heal nor-

mally, suggesting that the repair process in osteoporotic patients remains satisfactory [13].

Authors [12] reported that physical exercise increases skeletal bone mineral density (BMD) in a group of postmenopausal women and suggested that the lack of age-related physical exercise determines the decrease in bone mineral density.

## 4. Management of Osteoporotic Patients

This section addresses the considerations for the management of osteoporotic patients.

### 4.1. Clinical Examination

This process is directed towards detecting clinical features of the disease, as shown in the following sections.

#### 4.1.1. Clinical Signs

- Decreased thickness and height of the alveolar ridges, resulting in a decrease in vertical dimension [15] [16].

At the mandibular level, this induces the appearance of a knife-edge ridge as well as the impression of a long ascending branch due to the reduction in height of the horizontal branch [16].

- Reduction in the number of teeth: it has been shown that there is a statistically significant difference between the average number of missing teeth in osteoporotic patients (=14.4) compared to normal patients (=10.7) [1]. Osteoporosis is one of the factors responsible for tooth loss. This loss is linked to inflammation and demineralization of the bone around the tooth. The remaining teeth flatten and the jaw atrophies, giving the impression that the teeth are longer and more widely spaced. Bone resorption in the jaws and maxilla increases with edentulism. The distance between the chin and the nose shortens and the teeth move backward, eventually changing the appearance of the elderly person.
- Tooth mobility: due to significant resorption of the alveolar bone.
- Pain related to the presence of craniofacial disharmony: not always.
- Low muscle tone: Early tooth loss is responsible for atrophy of the masticatory muscles, leading to a deterioration in muscle tone and a loss of power, strength, endurance, and agility. The latter promotes the worsening of osteoporosis by understimulating the periosteum [17].

#### 4.1.2. Radiological Signs

At the orofacial level, some research is conducted on retroalveolar [18] or panoramic [19] radiographs. The methodology includes either the development of an index or computer processing with bone density assessment using a gray scale:

Cheng *et al.* [20] worked on a panoramic mandibular index (PMI). This index uses the thickness of the mandibular cortex assessed on a dental panoramic image, vertical to the mental foramen.

Tezal *et al.* (2000) [21] used the existing relationship between bone density and a vertebral index. They investigated the possibility of assessing the approximate osteoporotic risk of a patient with a large gap in the dental office. The results of this study demonstrated the relationship between the vertebral index and the number of remaining teeth but provided little information on the method used in analyzing the panoramic radiograph. They studied the "feasibility" of using dental panoramic images as a tool to prevent bone density changes in osteoporosis. They suggested using the gonial region (the site of the first signs of decalcification). To do this, the X-rays are digitized and the average gray saturation is assessed. A density decrease of 30% to 60% is commonly described as a starting point for detecting osteopenia. The authors showed that a 5.3% decrease can be detected using this method. Therefore, there are currently many avenues of research that are gaining interest [21].

According to Balshi *et al.* [11], from the Department of Maxillofacial Radiology at Hiroshima University, to demonstrate that panoramic dental X-rays can be used to screen for low bone mineral density (BMD) in postmenopausal women, he followed 3160 postmenopausal women who showed no symptoms of osteoporosis. The participants were divided into two groups: 157 had undergone a hysterectomy or oophorectomy, or had taken estrogen, while the others had neither history. Panoramic dental X-rays were taken of all participants and their cortical bone status and jaw width were assessed based on the X-rays. The results indicated that women with eroded cortical bone should undergo more extensive BMD testing.

Currently, screening questionnaires are heavily relied upon to identify women requiring more accurate BMD testing. The new study revealed that dental X-rays were just as effective as the screening questionnaire.

Assessing cortical bone status based on dental X-rays identified 87% of women with spinal osteoporosis in the group without a history of hysterectomy, oophorectomy, and estrogen therapy. This proportion was 80% in the other group. The questionnaire, for its part, would detect 87% of osteoporosis cases in women without a history of osteoporosis and 72% of cases in women with a history of osteoporosis.

According to Wactawski-Wende *et al.* [22], dental radiographs are not as specific as screening questionnaires, especially if postmenopausal women are not well informed about osteoporosis or have little interest in the problem. However, since panoramic radiographs to diagnose dental and jaw problems are part of common clinical practice worldwide, dentists could also examine the condition of the mandibular cortical bone and recommend BMD testing for women who need it.

The relationship between oral signs and osteoporosis was studied to assess the possibility of using these as indicators of postmenopausal osteoporosis. Women between 50 and 70 years of age were evaluated, 14 normal women and 21 osteoporotic women. Signs of osteoporosis consisted of thoracic fractures demonstrated

on lateral chest X-rays. Oral signs included the number of residual teeth, mandibular cortex width, alveolar bone resorption, and morphological classification of the inferior cortex on dental X-rays (**Figure 2**). There were no significant differences in any of the mandibular measurements between normal and osteoporotic subjects [23].

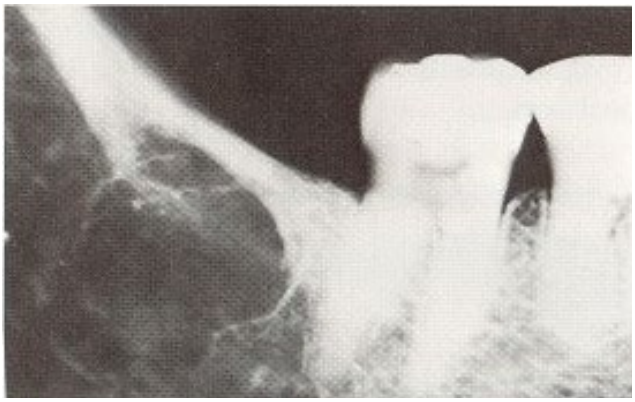


**Figure 2.** Retroalveolar radiograph showing bone resorption at the level of the mandibular alveolar ridge (M. SIDQUI).

In a tomographic study [6], it was shown that there was a significant decrease in total bone mineral density as well as trabecular mineral density in the partially edentulous mandibles of a group of rats after 12 weeks of ovariectomy. They observed a significant change in the mandibular bone trabeculations, which became loose and disconnected and decreased in number.

Zhu *et al.* [6] demonstrated the existence of alterations in the microarchitecture of the mandibular trabeculations of rats that had undergone ovariectomy. They concluded that the size of the narrow spaces and the shape of the trabeculations in the mandible could be a sign of osteoporotic involvement in the long bone and thus a sign of skeletal osteoporosis.

A cortical thickness at the gonion of less than 1 mm has been shown to be an indicator of decreased skeletal bone mineral density [6].

#### 4.1.3. Biological Signs

In general, the measurement of the elements of phospho-calcium metabolism allows for the initial ruling out of potential differential diagnoses. It includes:

- A blood test of total serum calcium, serum phosphorus, and sedimentation rate.
- A 24-hour urine calcium test.

Physiologically:

- Serum calcium: 100 mg/L.
- Serum phosphorus: 30 to 40 mg/L.
- Serum calcium: 150 to 250 mg/24 hours.
- Phosphaturia: linked to diet, for 1 g of ingested phosphorus, it is 600 mg/24 hours.

Biological markers characterize the activity of the bone cell pool and allow the detection of a potential imbalance in bone remodeling. Generally, indicators of collagen metabolism are used in the study of osteoporosis and hormone therapy in young, low-status patients [15] [24].

At the osteoblastic level:

- Alkaline phosphatase, average: 50 - 120 IU/L.
- Osteocalcin, average: 2 - 3 m mol/L.
- N- and C-terminal procollagen peptides, average: not determined.

At the osteoclastic level:

- Hydroxyproline: represents the best-known urinary measure of osteoclastic function. Its urinary excretion reflects the intensity of bone resorption. Its normal value is between 115 and 340 mmol/24 hours.
- Authors have used pyridonoline assays to quantify bone resorption (**Table 2**) [24].

**Table 2.** Normal values of biological markers (Reddy *et al.*) [24].

| Markers | Formation | Destruction | Normal Values |
|---|---|---|---|
| **Calcemia** | ++<br>+/– | +<br>+/– | 2.20 - 2.50 mmol/L |
| **Phosphorus** | ++/– | ++/– | 0.5 - 1.50 mmol/L |
| **Calciuria Spot**<br>**Phosphatase Acid TL** | ––<br>––<br>–+<br>–– | +<br>++++<br>+ | 5.8 +/– 2 mmol/ΔH<br>N. D |
| ***Phosphatase Alcaline*** | +<br>++++ | +<br>+/– | 50 - 120 UI/L |
| **Osteocalcn**<br>**Telopeptis NTX and Others** | +<br>++++<br>++++– | –<br>–<br>+++ | 2 - 3 mmol/L<br>N. D<br>N. D |
| ***PTH*** | +/– | +++ | 5 - 25 µmol/L |

Contribution of biology:

Mainly useful for eliminating:

- What is not osteoporosis:
  - Tumor, hypercalcemia, inflammation, etc.
  - Myeloma, hypercalcemia, peak, etc.
- What is endocrinopathy:
  - Hyperparathyroidism, hypercalcemia, PTH, etc.
  - Osteomalacia, vitamin D.
  - Others, thyroid or adrenal pathology.

According to Reddy *et al.* [24], there are no specific blood or urinary abnormalities in postmenopausal osteoporosis, but laboratory tests are essential for differential diagnosis to rule out other osteopathies, particularly osteomalacia (OM), ma-

lignant osteopathies, and primary hyperparathyroidism (HPT1), all of which can present with fragility fractures similar to those of osteoporosis.

- The sedimentation rate is normal. It is accelerated in cases of myeloma or bone metastases.
- Serum calcium is normal but may be low in cases of OM, elevated in cases of HPT.
- Serum phosphorus is normal but may be low in cases of HPT1 and OM.
- Serum alkaline phosphatase is normal but elevated in cases of OM.
- Serum parathyroid hormone is normal but may be elevated in cases of HPT1 or OM (due to secondary hyperparathyroidism).
- 25(OH) vitamin D is normal but may be low in cases of OM.
- Urinary calciuria and hydroxyprolinuria are within normal limits in postmenopausal osteoporosis. More sensitive and specific markers of bone turnover (serum osteocalcin, bone alkaline phosphatase, urinary free pyridinoline, collagen telopeptides) can help better assess the rate of bone loss and the effectiveness of treatments.
- A decrease in 25(OH)D, associated with an increase in parathyroid hormone levels, is common in elderly patients exposed to calcium and vitamin D deficiencies and at risk of femoral neck fracture.

#### 4.1.4. Histological Signs

The microscopic bone architecture reveals characteristic alterations, primarily assessed through specific histomorphometry technique.

**1) Qualitative and Quantitative Histomorphometry**

The microscopic appearance of osteoporosis is characteristic (**Figure 3**). It includes the following elements [15]:

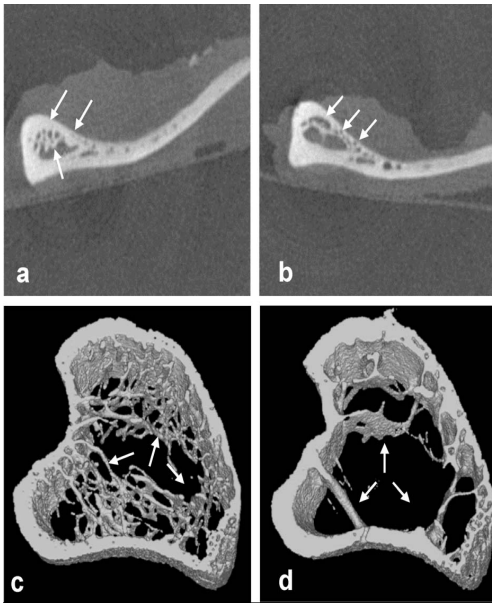



**Figure 3.** Comparison between healthy mandibular bone (right) and osteoporotic mandibular bone (left) [25].

- Thinning of the cortical bones.
- Atrophy of the splints.
- Scarcity of osteoblasts.
- Absence of osteoid margins.
- Hematopoietic tissue tends to shrink.
- Bone marrow that becomes adipose.

Histomorphometry studies both bone quantity and its architectural quality.

**2) Kinetic Histomorphometry**

Histomorphometry is the only technique that assesses the rate of bone remodeling as well as the number of BMUs (bone remodeling units) in the two weeks preceding the biopsy. It also allows for the assessment of bone remodeling kinematics using tetracycline markers. This examination is useful for validating the effect of a therapy on bone remodeling and, in rare cases, confirms complex diagnoses (such as osteoporosis, osteomalacia, and multiple myeloma) [15].

## 4.2. Drug Interactions

Drug co-administration demands interaction evaluation, the primary clinical concerns are outlined below.

### 4.2.1. Antiresorptives and Antibiotics

**Bisphosphonates and Antibiotics:**

One study investigated the effect of co-administering doxycycline and bisphosphonates [26].

Alendronate and/or doxycycline significantly inhibit most inflammatory mediators that cause periodontal tissue destruction; the combination of these two agents has a more effective benefit in the treatment of periodontal disease. Hormone replacement therapy and antibiotics [27]:

Enzymatic inhibitors such as rifampicin, barbiturates, antiepileptics, and griseofulvin may reduce or even reverse the action of estrogen-progesterone.

Concomitant administration of a systemic retinoid increases estrogen-progesterone inactivation.

Various medications may increase estrogen-progesterone (EP) toxicity: troleandomycin and cyclosporine combined with EPs may cause cholestatic hepatitis. Inhibitors such as metronidazole may increase the side effects of EPs (fluid and sodium retention, hypertension, venous thrombosis, diabetic effects, etc.).

EPs reduce the hydroxylation of certain medications such as tricycline antidepressants and theophylline. The dosage of these should be reduced by 30% at the beginning of treatment, otherwise overdose may occur. Combining OPs with mild antidiabetics is not recommended. In any case, diabetes and thrombosis already impose restrictions on the prescription of an OP.

### 4.2.2. Antiestrogens and Antibiotics

**Calcitonin and Antibiotics:**

There are no drug interactions with calcitonin in the literature [28].

#### 4.2.3. Calcium/Vitamin D and Antibiotics

The results of a review of 15 trials conducted on 1806 subjects followed for two years or more indicate that calcium is slightly more effective than a placebo in slowing bone loss in people with osteoporosis. In terms of prevention, the most recent studies and reviews contribute to establishing the effectiveness of the calcium and vitamin D combination for postmenopausal women. Research is now increasingly focused on other factors that may increase calcium absorption or slow calcium loss from bone. In this regard, it has recently been established that calcium supplementation enhances the beneficial effect of protein supplementation, increasing bone strength in older adults. It is also believed that adequate calcium intake in premenopausal women may help prevent osteoporosis. Today, calcium supplementation no longer plays an exclusively preventative role, as it is now part of medical treatment for osteoporosis, often in conjunction with vitamin D supplementation and hormone therapy. The combination of calcium and ipriflavone has also been shown to be effective in the prevention and treatment of osteoporosis [23] [29].

**Precautions:**

**Caution:**

**Renal Impairment or Hyperparathyroidism:**

Consult a physician before taking a calcium supplement. Kidney stones: The recommendation to reduce dietary calcium intake in people with kidney stones is unjustified, as recent data suggest that consuming foods rich in calcium may actually have a protective effect. Although the link between calcium supplement consumption and kidney stone formation is not supported by research, some experts recommend avoiding them.

Prostate cancer: There is a scientific hypothesis that calcium lowers vitamin D levels, one of the natural protective factors against prostate cancer. Epidemiological studies suggesting a possible link between high calcium intake and the incidence of this disease have produced conflicting results. Although some studies suggest a link between dairy product consumption and prostate cancer, other studies do not support this link or indicate that the evidence is too weak to draw definitive conclusions about the existence of this effect. Other studies indicate that if there is a link between calcium consumption and prostate cancer, it would be rather weak and would depend on an intake considerably exceeding the recommended nutritional intake.

Furthermore, it has recently been hypothesized that milk, rather than calcium, may be responsible for this effect, due to its estrogen, protein, and cysteine content. It has been noted that Asian men, who obtain their calcium primarily from soy and various vegetables rather than dairy products, have a lower incidence of prostate cancer than Westerners.

**Tolerable Upper Intake Level:**

Canadian and American medical authorities have set the Tolerable Upper Intake Level at 2500 mg per day. This is the highest daily amount that can be taken continuously without likely risk of adverse effects. Experts estimate the toxic dose

of calcium at 5 g per day, but consider that consuming more than 20 g per day is enough to cause hypercalcemia. Contraindications:

In cases of sarcoidosis (a rare disease, somewhat similar to tuberculosis, primarily affecting the skin and lymph nodes), calcium supplementation may cause hypercalcemia.

**Adverse Effects:**

Gastrointestinal irritation, belching, intestinal gas, constipation.

- Although no scientific data support this claim, calcium carbonate has been reported to cause constipation.

**Interactions:**

- **With Herbs or Supplements:**

- The absorption of iron and zinc may be hindered by calcium. It is recommended to leave a two-hour interval between taking calcium supplements and these minerals.
- It was long thought that taking calcium and magnesium supplements together could inhibit the absorption of one or the other of these minerals. Experts now believe that while this antagonism exists, it has no measurable clinical effect.

- **With Medications:**

- Bisphosphonates (such as alendronic acid). These osteoporosis medications inhibit osteoclast activity: they should be taken two hours before or after a calcium supplement.
- Amiloride. This diuretic and hypotensive medication reduces urinary calcium excretion.
- Atenolol. Calcium appears to lower blood levels of this beta-blocker.
- Gentamicin. Calcium appears to exacerbate the toxicity of this antibiotic.
- Quinolone antibiotics and tetracycline derivatives. Calcium appears to decrease the absorption of these antibiotics. Take two hours apart.

Tetracycline must first bind to the free $Ca^{2+}$ chelate in the serum, but ossification is no longer possible; the reaction does not occur in the mineral portion of the bone but in the collagen protein structure.

#### 4.2.4. Antiresorptives and Anti-Inflammatories

**Bisphosphonates and Anti-Inflammatory Drugs:**

In corticosteroid-induced osteoporosis, due to the interaction between the effects of corticosteroid treatment and those of the disease itself. The pathology of corticosteroid-induced osteoporosis and fracture development is complex; this topic actually deserves a separate, more detailed discussion based on limited data regarding the effect of different treatments on fracture risk in women treated with glucocorticoids. The current preference is for treatment with alendronate or residronate. Since systemic glucocorticoid use is itself a significant risk factor, cost-effective treatment is possible in women who, apart from glucocorticoid use, have other risk factors such as advanced age and low BMD, which are less prevalent than in postmenopausal osteoporosis [10] [30].

The prevention of steroid-induced osteoporosis justifies the combination of vitamin-calcium supplementation and bisphosphonate therapy when the prednisolone dose is greater than 7.5 mg/day [31].

Patients with cirrhosis frequently have osteoporosis, which is characterized by decreased collagen matrix synthesis and decreased mineralization, and is multifactorial. Cholestasis, IGF-1 deficiency, hypogonadism, vitamin D deficiency, malnutrition, and immobility play an important role in the genesis of bone pathology in these patients. Glucocorticoids administered after transplantation inhibit osteopathic activity and increase bone resorption.

These cumulative effects worsen pre-existing osteoporosis. Several studies have shown that bone mass decreases three to six months after transplantation. This decrease is related to steroid administration, but other medications, including cyclosporine and tacrolimus, may have an effect on bone mass. It is therefore not surprising to see a high rate of spontaneous fractures (especially vertebral compression fractures) during this period, ranging from 25 to 65% of patients. It is therefore important to perform a complete bone assessment in order to initiate treatment if necessary.

Regular monitoring (annual mineralometry) is essential. Treatment may include calcium and vitamin D supplements, hormone replacement (estrogen, progesterone), and bisphosphonates. Collaboration with a doctor specializing in bone diseases is important. Hormone replacement therapy and anti-inflammatories [30]:

The combination of hormone replacement and corticosteroid treatment is always possible (see the section on bisphosphonates and ATI: treatment of cirrhosis).

#### 4.2.5. Antiestrogens and Anti-Inflammatories

**Calcitonin and Anti-Inflammatories:**

No data are known regarding this medication [31].

#### 4.2.6. Other Medications

**Raloxifene:**

The available evidence does not support the use of raloxifene for fracture prevention in women treated with glucocorticoids [29].

**Teriparatides:**

No effect of this treatment on fracture risk in women treated with glucocorticoids has been demonstrated [29].

**Strontium Ranelate:**

To date, there are no published studies on strontium ranelate in osteoporosis. Cortisone [29].

#### 4.2.7. Calcium, Vitamin D, and Anti-Inflammatories

Patients receiving systemic glucocorticoid therapy experience reduced calcium absorption and increased calcium excretion, which can cause secondary hyperparthyoidism. Therefore, in these patients, it is always advisable to administer a calcium

and vitamin D supplement. In this case, the use of hydroxylated vitamin D derivatives or a higher dose of vitamin D is recommended [30].

The combination of calcium and vitamin D with corticosteroid treatment is always possible (see the section on bisphosphonates and ATIs for the treatment of cirrhosis) [30] [32].

#### 4.2.8. Interaction with Analgesics

**Antiresorptives and Analgesics** [26] [31]**:**

- Bisphosphonates and analgesics:

  There are no specific interactions for either paracetamol or dextroproxyfen.
- Hormone replacement therapy and analgesics:

  There are no interactions to report.

**Antiestrogens and Analgesics** [26] [31]**:**

- Calcitonin and analgesics:

  There are no specific interactions for either paracetamol or dextroproxyfen.

## 5. Conclusions

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

In the first chapter, we focused on diagnosis, the different etiological forms, risk factors, and preventive and curative treatment of the disease.

Prevention based on bone densitometry allows for early detection of the disease and the implementation of appropriate behavior.

Hormone replacement therapy and bisphosphonates are the main drug treatments prescribed for these patients.

In the second chapter, we studied, in the first part, the impact of osteoporosis on the oral cavity. Namely:

- Maxillary bases: The most affected sites are the anterior maxilla at the maxillary level, the gonion, the inferior border of the mandible, and the condyle at the mandibular level.
- Alveolar bone.
- Tooth loss.
- Bone healing.

In the second part of the second chapter, we focused on studying the various drug interactions between medications prescribed in dentistry (antibiotics, anti-inflammatories, and analgesics) and in osteoporotic patients, who are often taking multiple medications. This requires careful attention, particularly during the interview.

The third chapter, based on a series of studies, addressed the management of osteoporotic patients in dentistry. Several consequences must be considered: these elderly and fragile patients must be treated with even greater gentleness and sensitivity, both mentally and physically, particularly during extraction procedures.

## Conflicts of Interest

The authors declare no conflicts of interest.

## References


[1] Ensrud, K.E. and Crandall, C.J. (2024) Osteoporosis. *Annals of Internal Medicine*, **177**, ITC1-ITC16. https://doi.org/10.7326/aitc202401160

[2] Willers, C., Norton, N., Harvey, N.C., Jacobson, T., Johansson, H., Lorentzon, M., *et al.* (2022) Osteoporosis in Europe: A Compendium of Country-Specific Reports. *Archives of Osteoporosis*, **17**, Article No. 23. https://doi.org/10.1007/s11657-021-00969-8

[3] Walker, M.D. and Shane, E. (2023) Postmenopausal Osteoporosis. *New England Journal of Medicine*, **389**, 1979-1991. https://doi.org/10.1056/nejmcp2307353

[4] Vilaca, T., Eastell, R. and Schini, M. (2022) Osteoporosis in Men. *The Lancet Diabetes & Endocrinology*, **10**, 273-283. https://doi.org/10.1016/s2213-8587(22)00012-2

[5] Bandrivsky, Y., Bambuliak, A., Bandrivska, O., Maika, I., Dutko, K. and Kuchyrka, L. (2024) Pharmacological Correction of the Activity of Bone Remodelling Markers in the Oral Fluid of Patients with Generalised Periodontitis Depending on Blood Type. *Pharmacia*, **71**, 1-6. https://doi.org/10.3897/pharmacia.71.e114268

[6] Zhu, L., Zhou, C., Chen, S., Huang, D., Jiang, Y., Lan, Y., *et al.* (2022) Osteoporosis and Alveolar Bone Health in Periodontitis Niche: A Predisposing Factors-Centered Review. *Cells*, **11**, Article 3380. https://doi.org/10.3390/cells11213380

[7] Emami, Z., Zamani, S., Kiany, F., Khojastepour, L. and Zamani, A. (2022) Evaluation of the Association between Osteoporosis and Periodontitis in Postmenopausal Women: A Clinical and Radiographic Study. *Dental Research Journal*, **19**, Article 41. https://doi.org/10.4103/1735-3327.346399

[8] Bandrivsky, Y., Bandrivska, O., Goncharuk-Khomyn, M., *et al.* (2024) Morphometric Features of Periodontal Phenotype and Anthropometric Parameters of the Maxillary Central Incisor in Patients with Generalized Periodontitis and Various Blood Types. *Journal of International Dental and Medical Research*, **17**, 168-175.

[9] Bandrivsky, Y., Bandrivska, O., Gnid, R., Minko, L. and Shevchuk, M. (2019) Indicators of Markers of Bone Metabolism in Patients with Generalized Periodontitis Depending on Blood Group. *Archives of the Balkan Medical Union*, **54**, 72-77. https://doi.org/10.31688/abmu.2019.54.1.10

[10] Herbert, A.M., Dean, M.N., Summers, A.P. and Wilga, C.D. (2022) Biomechanics of the Jaws of Spotted Ratfish. *Journal of Experimental Biology*, **225**, jeb243748. https://doi.org/10.1242/jeb.243748

[11] Balshi, T.J., Balshi, S.F. and Wolfinger, G.J. (2007) The Evolution of Advanced Prosthodontic Care: A 30-Year Patient Report. *Journal of Prosthodontics*, **16**, 43-49. https://doi.org/10.1111/j.1532-849x.2006.00153.x

[12] Tsyhykalo, O.V., Kuzniak, N.B., Dmytrenko, R.R., Perebyjnis, P.P., Bernik, N.V., Krynychnykh, H.I., *et al.* (2022) Peculiarities of the Human Maxilla Morphogenesis. *Wiadomości Lekarskie*, **75**, 2339-2346. https://doi.org/10.36740/wlek202210105

[13] Hildebolt, C.F. (1997) Osteoporosis and Oral Bone Loss. *Dentmaxillofacial Radiology*, **26**, 3-15. https://doi.org/10.1038/sj.dmfr.4600226

[14] Payne, J.B., Stoner, J.A., Nummikoski, P.V., Reinhardt, R.A., Goren, A.D., Wolff, M.S., *et al.* (2007) Subantimicrobial Dose Doxycycline Effects on Alveolar Bone Loss in Post-Menopausal Women. *Journal of Clinical Periodontology*, **34**, 776-787. https://doi.org/10.1111/j.1600-051x.2007.01115.x

[15] Nasu, M., Amano, Y., Kurita, A. and Yosue, T. (1998) Osseointegration in Implant-embedded Mandible in Rats Fed Calcium-Deficient Diet: A Radiological Study. *Oral Diseases*, **4**, 84-89. https://doi.org/10.1111/j.1601-0825.1998.tb00262.x

[16] Hong, S.W., Lee, J. and Kang, J. (2023) Associations between Oral Health Status and Risk of Fractures in Elder Adults. *Scientific Reports*, **13**, Article No. 1361. https://doi.org/10.1038/s41598-023-28650-9

[17] Ohishi, T. and Matsuyama, Y. (2018) Minodronate for the Treatment of Osteoporosis. *Therapeutics and Clinical Risk Management*, **14**, 729-739. https://doi.org/10.2147/tcrm.s149236

[18] Li, Y., Ling, J. and Jiang, Q. (2021) Inflammasomes in Alveolar Bone Loss. *Frontiers in Immunology*, **12**, Article 691013. https://doi.org/10.3389/fimmu.2021.691013

[19] Belluci, M.M., de Molon, R.S., Rossa Jr., C., Tetradis, S., Giro, G., Cerri, P.S., *et al.* (2020) Severe Magnesium Deficiency Compromises Systemic Bone Mineral Density and Aggravates Inflammatory Bone Resorption. *The Journal of Nutritional Biochemistry*, **77**, Article 108301. https://doi.org/10.1016/j.jnutbio.2019.108301

[20] Cheng, C., Chen, L. and Chen, K. (2022) Osteoporosis Due to Hormone Imbalance: An Overview of the Effects of Estrogen Deficiency and Glucocorticoid Overuse on Bone Turnover. *International Journal of Molecular Sciences*, **23**, Article 1376. https://doi.org/10.3390/ijms23031376

[21] Tezal, M., Wactawski-Wende, J., Grossi, S.G., Ho, A.W., Dunford, R. and Genco, R.J. (2000) The Relationship between Bone Mineral Density and Periodontitis in Postmenopausal Women. *Journal of Periodontology*, **71**, 1492-1498. https://doi.org/10.1902/jop.2000.71.9.1492

[22] Wactawski-Wende, J., Grossi, S.G., Trevisan, M., Genco, R.J., Tezal, M., Dunford, R.G., *et al.* (1996) The Role of Osteopenia in Oral Bone Loss and Periodontal Disease. *Journal of Periodontology*, **67**, 1076-1084. https://doi.org/10.1902/jop.1996.67.10s.1076

[23] Krall, E.A. (2011) Osteoporosis and the Risk of Tooth Loss. *Clinical Calcium*, **16**, 287-290

[24] Reddy, M.S., Geurs, N.C. and Gunsolley, J.C. (2012) Periodontal Host Modulation with Antiproteinase, Anti-Inflammatory, and Bone-Sparing Agents. A Systematic Review. *Annals of Periodontology*, **8**, 12-37. https://doi.org/10.1902/annals.2003.8.1.12

[25] Mattson, J.S., Cerutis, D.R. and Parrish, L.C. (2018) Osteoporosis: A Review and Its Dental Implications. *Compendium of Continuing Education in Dentistry*, **23**, 1001-1014.

[26] Jeffcoat, M. (2005) The Association between Osteoporosis and Oral Bone Loss. *Journal of Periodontology*, **76**, 2125-2132. https://doi.org/10.1902/jop.2005.76.11-s.2125

[27] Dervis, E. (2005) Oral Implications of Osteoporosis. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*, **100**, 349-356. https://doi.org/10.1016/j.tripleo.2005.04.010

[28] Amorim, M.A.L., Takayama, L., Jorgetti, V. and Pereira, R.M.R. (2006) Comparative Study of Axial and Femoral Bone Mineral Density and Parameters of Mandibular Bone Quality in Patients Receiving Dental Implants. *Osteoporosis International*, **17**, 1494-1500. https://doi.org/10.1007/s00198-006-0131-0

[29] Yu, B. and Wang, C. (2022) Osteoporosis and Periodontal Diseases—An Update on Their Association and Mechanistic Links. *Periodontology 2000*, **89**, 99-113. https://doi.org/10.1111/prd.12422

[30] Koth, V.S., Salum, F.G., de Figueiredo, M.A.Z. and Cherubini, K. (2021) Repercus-

sions of Osteoporosis on the Maxillofacial Complex: A Critical Overview. *Journal of Bone and Mineral Metabolism*, **39**, 117-125. https://doi.org/10.1007/s00774-020-01156-4

[31] Penoni, D.C., Fidalgo, T.K.S., Torres, S.R., Varela, V.M., Masterson, D., Leão, A.T.T., *et al.* (2017) Bone Density and Clinical Periodontal Attachment in Postmenopausal Women: A Systematic Review and Meta-Analysis. *Journal of Dental Research*, **96**, 261-269. https://doi.org/10.1177/0022034516682017

[32] Sidiropoulou-Chatzigiannis, S., Kourtidou, M. and Tsalikis, L. (2007) The Effect of Osteoporosis on Periodontal Status, Alveolar Bone and Orthodontic Tooth Movement. A Literature Review. *Journal of the International Academy of Periodontology*, **9**, 77-84.