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Understanding Osteoporosis: Pathophysiology, Risk Factors, Diagnosis, and Management

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Abstract

Osteoporosis is a systemic skeletal disease characterized by low bone mineral density (BMD) and deterioration of bone architecture, resulting in reduced bone strength and, consequently, increased susceptibility to fractures which poses a significant public health concern worldwide, particularly in aging populations [1]. The health-economic impact of vertebral and hip fractures has been extensively explored and it is well known that these fractures are associated with morbidity/disability and increased mortality; they also account for a substantial portion of the direct fracture costs. This review aims to provide a comprehensive overview of osteoporosis, including its pathophysiology, risk factors, diagnostic approaches, and management strategies. By elucidating the multifaceted nature of this condition, healthcare providers can better identify individuals at risk, implement preventive measures, and optimize treatment to reduce the burden of osteoporotic fractures.

Keywords

Osteoporosis, Bone Mineral Density, Fractures, Risk Factors, Diagnosis, Management, FRAX (Fracture Risk Assessment Tool), Trabecular Bone Score (TBS)

1. Introduction

Osteoporosis is a silent disease characterized by reduced bone strength, predisposing individuals to fragility fractures, which significantly impact morbidity, mortality, and quality of life. Per World Health Organization (WHO) definitions, osteoporosis is bone mineral density (BMD) 2.5 SD or more below that of young normal individual (T score −2.5 or less). Osteopenia (or low bone mass) is BMD scores between 1 and 2.5 SD below young normals (T score between −1 and −2.5). Severe osteoporosis is bone mineral density (BMD) 2.5 SD or more

below that of young normal individual in the presence of one or more fragility fractures. These definitions are applicable to postmenopausal women and men older than 50 years old. From pathology standpoint, osteoporosis is a systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility. Established osteoporosis is occurrence of minimal trauma fracture of any bone (fragility fracture). Fragility (Low or minimal trauma) fracture is non pathological fracture that occurs from a fall from standing height or less (feet, hands and skull excluded) [2] [3]. Despite its prevalence and clinical significance, osteoporosis remains underdiagnosed and undertreated resulting in decrease in quality of life and an increase in premature mortality, disability, and financial burden [4] [5] [6] [7]. More than 2 million osteoporosis-related fractures occur annually in the United States. Approximately 1 in 2 white women and 1 in 5 men will experience an osteoporotic-related fracture in their lifetime. By 2025, the burden in the country is projected to increase by almost 50% to more than 3 million fractures and US $253 billion per year highlighting the need for enhanced awareness and proactive management strategies. This review aims to consolidate current knowledge on osteoporosis, providing clinicians with a comprehensive resource for effective patient care [8].

2. Pathophysiology

Osteoporosis is due to imbalance between bone formation and bone resorption. Bone is continually remodeled throughout our lives (bone turnover). Bone remodeling occurs at discrete sites within the skeleton and proceeds in an orderly fashion, and bone resorption is always followed by bone formation, a phenomenon referred to as coupling. In women, bone mass peaks around the third decade of life and slowly decreases afterward. After menopause, the rate of bone loss is accelerated for 8 - 10 years. In men, accelerated bone loss starts late around age of 75. Various factors contribute to this dysregulation, including hormonal changes, genetic predisposition, nutritional deficiencies, and systemic illnesses. Disruption of the delicate interplay between osteoblasts and osteoclasts, the bone remodeling units, accelerates bone loss, ultimately predisposing individuals to fractures [9].

2.1. Risk Factors

Numerous risk factors influence an individual's susceptibility to osteoporosis and fracture. Age (≥50 years), female sex, white or Asian ethnicity, genetic factors, such as a family history of osteoporosis, cystic fibrosis, hypophosphatasia, Ehlers-Danlos, osteogenesis imperfecta, thin build or small stature (eg, body weight less than 127 lb [57.6 kg]), amenorrhea, late menarche, early menopause, postmenopausal state, physical inactivity or immobilization, use of certain drugs (eg, anti-epileptic drugs, systemic steroids, thyroid supplements, heparin, chemotherapeutic agents, insulin, proton pump inhibitors, androgen deprivation therapy), alcohol and tobacco use, androgen or estrogen deficiency and calcium and vitamin D deficiency contribute to skeletal fragility and fracture risk [10].

2.2. Diagnosis

Osteoporosis generally does not become clinically apparent until a fracture occurs. Two thirds of vertebral fractures are painless [11]. Screening for early detection of osteoporosis is essential for timely intervention and fracture prevention. Dual-energy X-ray absorptiometry (DXA) scan remains the gold standard for assessing BMD and diagnosing osteoporosis. USPSTF recommends screening women 65 year old or above without previous known fractures or secondary causes of osteoporosis and women less than 65 year old with 10 year fracture risk equal or greater than that of 65 white female (9.3% based on FRAX US risk factors) [12]. T-score value as within 1 standard deviation (SD) of the mean BMD value in a healthy young adult. Values lying farther from the mean are considered abnormal. T-score of −1 to −2.5 SD indicates osteopenia or low bone mass. T-score of −2.5 SD or less indicates osteoporosis. T-score of less than −2.5 SD with fragility fracture (s) indicates severe osteoporosis. Clinical risk assessment tools, such as FRAX® (Fracture Risk Assessment Tool) is used to determine a person's 10-year risk of major osteoporotic fractures and 10-year risk of hip fracture. FRAX score is calculated based on 12 factors namely age, sex, weight, height, prior fracture, parent's hip fracture, current smoking, steroid use, rheumatoid arthritis, secondary causes of osteoporosis, drinking of 3 or more alcoholic beverages per day and BMD [13] [14]. Trabecular bone score (TBS) is an assessment of how evenly or unevenly mineral is structurally distributed in trabecular bone. A TBS is generated from lumbar spine BMD images using software installed on a DXA machine. No additional scan time or radiation exposure is required. The TBS gray-scale texture model captures local differences in mineral concentrations, providing an index of bone microarchitecture that predicts fracture risk independent of BMD and FRAX® scores [15]. However, adipose tissues can generate artifact and interfere with the accuracy of TBS, thus limiting its generalizability [16].

2.3. Management

The management of osteoporosis encompasses lifestyle modifications, pharmacotherapy, and fracture prevention strategies. Lifestyle interventions, including weight-bearing exercise, adequate calcium and vitamin D intake, smoking cessation, and moderation of alcohol consumption, form the foundation of osteoporosis management. Furthermore, fall prevention strategies, including environmental modifications and balance training, play a crucial role in mitigating fracture risk in older adults. Pharmacological agents are indicated in patients with fragility fracture and patients with T-score ≤ −2.5 at the femoral neck or spine after appropriate evaluation to exclude secondary causes.

Patients with osteopenia or low bone mass (T-score between −1.0 and −2.5 at the femoral neck or spine) and have a 10 year probability of hip fracture equal to or more than 3% or a 10 year probability of major osteoporotic fracture equal to or more than 20% (FRAX score) require pharmacological treatment as well [17].

3. General Measures

3.1. Adequate Intake of Calcium and Vitamin D

Bone Health and Osteoporosis Foundation (BHOF) supports the Institute of Medicine's (IOM) calcium intake recommendations: 1000 mg/day for men aged 19 - 70 years and women aged 19 - 50 years; 1200 mg/day for women 51 years and older and men 71 years and older. The BHOF recommends a daily intake of 800 to 1000 units of vitamin D for adults aged 50 years and older. The Institute of Medicine Dietary Reference Intakes for vitamin D are 600 units daily until age 70 years and 800 units/day for adults age 71 years and older [18] [19].

3.2. Cessation of Smoking and Alcohol Abuse

Bone Health and Osteoporosis Foundation (BHOF) strongly recommends smoking cessation to support primary and secondary prevention of osteoporosis. Moderate alcohol intake has no known negative effect on bone and may even be associated with slightly higher bone density and lower risk of fracture in postmenopausal women. However, alcohol intake of more than two drinks a day for women or three drinks a day for men may be detrimental to bone health. It has been associated with reduced calcium absorption and increased risk for falls. Clinicians should identify patients at risk for chronic heavy drinking and/or binge drinking who require further evaluation and treatment [20] [21].

3.3. Weight-Bearing Exercise

Bone Health and Osteoporosis Foundation (BHOF) strongly endorses physical activity at all ages, both for fracture prevention and overall fitness. In childhood and adolescence, consistent weight-bearing and high-impact activities contribute to acquisition of optimal peak bone mass. Weight-bearing exercises (in which bones and muscles work against gravity with feet and legs bearing body weight) include walking, jogging, tai chi, stair climbing, dancing, and tennis [22].

3.4. Fall Prevention Strategies

Multiple studies have demonstrated the efficacy of therapeutic physical activity in reducing falls. A recent meta-analysis of RCTs investigating moderate-intensity multicomponent physical activity (aerobic, balance, and strength training) 3 times a week for 1 year or more reported significant fall reductions: 22% lower risk for falls and 26% lower risk for injurious falls. Risk of fractures was reduced by 16%, although the significance of this finding is weakened by the small number of fractures in the study ($p = 0.05$) [23].

4. Pharmacotherapy

4.1. Bisphosphonates (Alendronate, Risedronate, Ibandronate and Zoledronic Acid)

These agents are considered the first line of osteoporosis pharmacotherapy treat-

ment. Bisphosphonates inhibit osteoclastic activity; reducing bone resorption and turnover. The limited trial data available regarding long-term treatment with bisphosphonates has raised questions about the optimal length of treatment with these medications. This issue has become more important, given newly recognized complications of bisphosphonate use, including osteonecrosis of the jawand atypical (subtrochanteric or femoral shaft) femur fractures. It is recommended to consider 2 - 3 years of drug holiday after 5 years of oral and 3 years of IV bisphosphonate therapy [24] [25]. Other important side effects of bisphosphonates include upper GI symptoms and esophagitis especially if medication was not taking as prescribed, hypocalcemia and musculoskeletal pain especially in patients with low Vitamin D and acute phase response with flu like symptoms especially with IV forms. Bisphosphonates are contraindicated in patients with creatinine clearance less than 35.

4.2. Alendronate (Fosamax)

- Approved for the treatment of osteoporosis in men, in postmenopausal women, and in patients with glucocorticoid-induced osteoporosis.
- Dose for osteoporosis treatment is 70 mg/wk, to be taken sitting upright with a large glass of water at least 30 minutes before eating in the morning.
- Alendronate reduces incidence of spine and hip fractures by about 50% over 3 years in patients with prior vertebral fracture and in patients who have hip T-scores diagnostic of osteoporosis (≤−2.5) [26]. It reduces incidence of vertebral fractures by 48% over 3 years in patients without prior vertebral fracture.

4.3. Risedronate (Actonel)

- Approved for the treatment of osteoporosis in men, in postmenopausal women, and in patients with glucocorticoid-induced osteoporosis.
- 35 mg q week or 150 mg po q month. 5 mg po daily for osteoporosis prevention in patients on > 7.5 mg/day prednisone or equivalent.
- Compared with placebo, risedronate reduced incidence of vertebral fractures by 39%, hip fractures by 27%, and non-vertebral fractures by 22% in a meta-analysis conducted by Barrionuevo *et al.* in 2019 [27]. Significant risk reduction occurred within 1 year of treatment in patients with a prior vertebral fracture [13].

4.4. Ibandronate (Boniva)

- Approved for treatment treatment of osteoporosis in postmenopausal women,
- Dose 150 mg orally once a month and is also available as an intravenous formulation that is given 3 mg every 3 months.
- It reduces incidence of vertebral fractures by about 33% - 50% over 3 years but does not reduce risk of non-vertebral fracture (hip/nonhip) [28].

4.5. Zoledronic Acid (Reclast)

- Dose 5 mg IV yearly intravenous infusion
- Approved for the treatment of osteoporosis in men, in postmenopausal women, and in patients with glucocorticoid-induced osteoporosis.
- Most potent bisphosphonate available. It increases BMD at the spine by 4.3% - 5.1% and the hip by 3.1% - 3.5%, as compared with placebo. Over 3 years, it reduces the incidence of spine fractures by 70%, hip fractures by 41%, and nonvertebral fractures by 25%. A similar effect on vertebral fractures has been shown in men. A 2012 randomized, 2-year trial of men with osteoporosis found that once-yearly zoledronic acid infusions significantly decreased the risk of new morphometric vertebral fractures by 67% [29] [30].

5. Monoclonal Antibodies (Denosumab and Romosozumab)

5.1. Denosumab (Prolia)

This is a monoclonal antibody whichbinds the cytokine nuclear factor-kappa B ligand (RANKL), which is essential for formation, function and survival of osteoclasts which are responsible for bone resorption. It should be considered in certain patients with renal insufficiency, as impaired renal function does not significantly affect the metabolism or excretion of the drug. It reduces bone resorption by inhibiting the development of osteoclasts. It circulates in the blood for up to 9 months after subcutaneous injection; once cleared from the circulation, bone resorption transiently but dramatically increases, resulting in an abrupt decline in bone mineral density and, in some cases, vertebral fractures. It is recommended to transition patients to other antiresorptive treatment after discontinuing denosumab. The most statistically significant adverse events associated with denosumab include eczema, injection site reaction, hypocalcemia, and increased risk of infections, especially of the skin. There are case reports of osteonecrosis of the jaw with denosumab use, as well as atypical femoral fractures. Because of the risk of hypocalcemia, BHOF guidelines recommend evaluating and repleting calcium and vitamin D before starting treatment. Furthermore, monitoring serum calcium, phosphorus, magnesium, and for signs of infection should be a regular part of follow-up care while a patient is on denosumab [31] [32] [33] [34]. In January 2024, FDA (Food and Drug Administration) issued boxed warning with regard severe hypocalcemia in advanced Chronic Kidney Disease patients (eGFR <30) including dialysis patients using denosumab.

5.2. Romosozumab (Evenity)

This is a monoclonal antibody that binds with and inhibits sclerostin. Sclerostin, the product of the *SOST* gene has primarily been studied for its profound impact on bone mass. By interacting with LRP5 and LRP6, the glycoprotein suppresses the propagation of Wnt signals to β-catenin and thereby suppresses new bone formation [35]. Thus, Romosozumab both increases bone formation and decreases bone resorption. It was approved by the FDA in 2019 for treatment of

osteoporosis in postmenopausal women who are at high risk for fracture. The dose is 210 mg subcutaneous monthly for 12 months.

It has been shown to reduce vertebral fracture rates in postmenopausal women with osteoporosis. In postmenopausal women with osteoporosis, romosozumab was associated with a lower risk of vertebral fracture than placebo at 12 months and, after the transition to denosumab, at 24 months. The lower risk of clinical fracture that was seen with romosozumab was evident at 1 year. In the Fracture Study in Postmenopausal Women With Osteoporosis (FRAME), a phase 3 randomized trial in 7180 postmenopausal women who had a T score of −2.5 to −3.5 at the total hip or femoral neck, 1 year of treatment with romosozumab reduced vertebral fracture rates by 73% compared with placebo. A further reduction in vertebral fracture risk occurred in the second year following transition to denosumab.

Side effects: Romosozumab received FDA approval with a boxed warning stating that it may increase risks for myocardial infarction, stroke, and cardiovascular (CV) death. It should not be taken by women who experienced a stroke or CV event in the previous year. Romosozumab may cause hypocalcemia, and therefore, hypocalcemia must be corrected before treatment. In studies, romosozumab has been associated with hypersensitivity reactions, including angioedema, erythema multiforme, dermatitis, rash, and urticaria. Romosozumab has been associated with rare cases of AFF and ONJ (fewer cases than denosumab) [36] [37].

6. Parathyroid Hormone Analogues (Teriparatide, Abaloparatide)

Parathyroid hormone (PTH) regulates calcium homeostasis. Constant high exposure to PTH causes bone resorption, while intermittent administration of exogenous recombinant PTH stimulates bone formation.

6.1. Teriparatide (Forteo)

Teriparatide is a synthetic fragment of human PTH that is approved by the FDA for treatment of osteoporosis in men and women at high risk for fracture (which is defined as a history of osteoporotic fracture, multiple risk factors for fracture, or failure/intolerance to other available osteoporosis therapy). It is approved to treat glucocorticoid-induced osteoporosis in men and women at high risk for fracture [38]. The FDA has approved an expanded indication for teriparatide for treatment of osteoporosis associated with sustained systemic glucocorticoid therapy (≥ 5 mg/day of prednisone). Forteo® is currently available as 20 μg daily subcutaneous injection.

Teriparatide reduces risk of vertebral fractures by 65% - 77%, and non-vertebral fractures by 35% - 53% in patients with osteoporosis, after an average of 18 months of therapy [39]. The VERO trial that compared teriparatide and risedronate in postmenopausal women with severe osteoporosis reported ~56% fewer

new vertebral fractures in the teriparatide group after 24 months [40]. It is important to follow teriparatide treatment with an antiresorptive agent, usually a bisphosphonate or denosumab, to maintain or further increase BMD.

Teriparatide is administered by 20 μg daily subcutaneous injection. When treatment is discontinued, bone loss can be rapid and alternative agents should be considered to maintain BMD.

Side effects of teriparatide include transient orthostatic hypotension, leg cramps, and nausea. Teriparatide transiently increases serum calcium which may predispose patients to digitalis toxicity. It should be used with caution in patients with active or recent kidney stones, hypercalcemia and hypercalcemic disorders, and/or cutaneous calcifications.

Until recently, teriparatide treatment was restricted to 2 years in response to elevated osteosarcoma seen in rodent studies. Increased osteosarcoma was not observed in humans during 15 years of post-marketing studies. As a result, the revised teriparatide label now states that use for more than 2 years during a patient's lifetime can be considered if a patient remains at or has returned to having a high risk for fracture.

Its use should be avoided in settings of increased risk for osteosarcoma: Paget's disease of the bone, prior radiation therapy involving the skeleton, open epiphyses (children and young adults), history of bone metastases or malignancies, unexplained elevated alkaline phosphatase, and hereditary disorders predisposing to osteosarcoma [39].

6.2. Abaloparatide (Tymlos)

Synthetic peptide analog of human PTH-related protein approved by the FDA for treatment of osteoporosis in postmenopausal women at high risk for fracture defined as a history of osteoporotic fracture, multiple risk factors for fracture, or failure/intolerance to other available osteoporosis therapy.

Abaloparatide reduces risk of new vertebral fractures by about 86% and non-vertebral fractures by about 43% in postmenopausal women with osteoporosis, after an average of 18 months of therapy [41]. In an extension study (ACTIVE-Extend) after 18 months of abaloparatide or placebo, the addition of 6 months of oral alendronate for a total of up to 24 months of therapy resulted in a relative risk reduction of radiographic spine fractures by 87%, non-vertebral fractures by 52%, and major osteoporotic fractures by 58% [42].

Abaloparatide is administered by 80 μg daily subcutaneous injection in the periumbilical area of the abdomen. When treatment is discontinued, bone loss can be rapid. An antiresorptive agent should be considered to maintain BMD. In December 2021, Black Box warning regarding increased risk of osteosarcoma, based on studies on rats, was removed; however, use of PTH analogues should be avoided in patients with increased risk of osteosarcoma, and use of the drug for more than 2 years during a patient's lifetime is not recommended.

Side effects of abaloparatide include leg cramps, nausea, and dizziness. Avoid

use in patients with increased risk of osteosarcoma (e.g., Paget's disease of bone, bone metastases, prior skeletal radiation). Patients with hypercalcemia, or a history of an unexplained elevated alkaline phosphatase or skeletal malignancy should not receive abaloparatide therapy. Abaloparatide may increase urinary calcium. It should be used with caution in patients with active or recent kidney stones because of the potential to exacerbate this condition. It is common practice to follow abaloparatide treatment with an antiresorptive agent, usually a bisphosphonate or denosumab, to maintain or further increase BMD.

7. Estrogen-Related Therapies (ET/HT, SERMs and Bazedoxifene/Conjugated Estrogens)

7.1. Estrogen Therapy/Menopausal Hormonal Therapy (ET/HT)

Estrogen/hormone therapy is approved by the FDA for prevention of osteoporosis and relief of vasomotor symptoms and vulvovaginal atrophy associated with menopause. Women with an intact uterus require HT (combined estrogen and progestin) to protect uterine lining. Women who have had a hysterectomy are treated with ET (estrogen alone).

The Women's Health Initiative (WHI) found that 5 years of oral HT (Prempro®) reduced incidence of clinical vertebral fractures and hip fractures by 34% and other osteoporotic fractures by 23% [43]. Meta-analysis sponsored by the Endocrine Society found that HT reduced fractures of the spine by 35%, hip by 28%, and non-vertebral skeleton by 22% [27].

ET/HT is available in a wide variety of oral and transdermal preparations that contain estrogen only, progestin only, and combination estrogen-progestin. ET/HT dosages include cyclic, sequential, and continuous regimens. When treatment is discontinued, bone loss can be rapid. Follow-on antifracture agents should be considered to maintain BMD.

Side effects and drug safety: Potential risks for women include biliary issues, breast cancer (with combined estrogen-progestin), endometrial hyperplasia/cancer (with inadequately opposed estrogen). Initial WHI data found elevated risk of myocardial infarction, stroke, pulmonary emboli, and deep vein thrombosis during 5 years of treatment with conjugated equine estrogen and medroxyprogesterone acetate (Prempro®) [44]. Subsequent analyses of WHI substudy data showed no increase in cardiovascular disease in women starting treatment within 10 years of menopause [45].

The North American Menopause Society (NAMS) and American Association of Clinical Endocrinologists (AACE)/American College of Endocrinology (ACE) recommend tailoring ET/HT formulation, dose, and route of administration to individual postmenopausal women. Risk-benefit profiles differ by patient age, time since menopause, and other factors [46] [47].

The Endocrine Society guidelines recommend ET/HT to prevent fractures in some high-fracture-risk postmenopausal women <60 years of age or <10 years past menopause who are experiencing vasomotor and/or climacteric symptoms

and cannot take bisphosphonates or denosumab [47].

When ET/HT use is considered solely for fracture prevention, the FDA recommends that approved non-estrogen treatments first be carefully considered.

The USPSTF recommends against the use of combined estrogen and progestin for the primary prevention of chronic conditions in postmenopausal persons. (D recommendation) The USPSTF recommends against the use of estrogen alone for the primary prevention of chronic conditions in postmenopausal persons who have had a hysterectomy. (D recommendation) [48].

7.2. Selective Estrogen Receptor Modulators (SERMs)

Raloxifene (Evista) is a SERM indicated for the treatment and prevention of osteoporosis in postmenopausal women. The usual dose is 60 mg given orally daily. It has been shown to increase the incidence of deep vein thrombosis, stroke, and hot flashes. It is recommended for postmenopausal women with osteoporosis and in need for treatment of prevention or treatment of breast cancer. Raloxifene reduces incidence of vertebral fractures by about 30% - 40% in patients with a prior vertebral fracture and by about 55% in patients without a prior vertebral fracture. Raloxifene does not reduce risk of non-vertebral fractures. Raloxifene increases risk for deep vein thrombosis to a degree similar to that observed with estrogen. It can increase hot flashes and cause leg cramps [17] [49].

7.3. Bazedoxifene/Conjugated Estrogens (Duavee)

The combination product of bazedoxifene, a SERM, and conjugated estrogens (CEs) is approved by the FDA for prevention of osteoporosis and treatment of vasomotor symptoms in postmenopausal women. Combining a SERM with CEs lowers the risk of uterine hyperplasia caused by estrogens. This eliminates the need for a progestin and its associated risks (e.g., breast cancer, myocardial infarction, venous thromboembolism).

In pivotal trials, this combination drug significantly increased mean lumbar spine BMD (treatment difference 1.51%) at 12 months compared to placebo in women who had been postmenopausal between 1 and 5 years. Treatment with conjugated estrogens/bazedoxifene also increased total hip BMD. The treatment difference in total hip BMD at 12 months was 1.21%. Side effects of conjugated estrogens/bazedoxifene include muscle spasms, nausea, diarrhea, dyspepsia, upper abdominal pain, oropharyngeal pain, dizziness, and neck pain. Because this product contains estrogen, it is approved with the same Boxed Warning and other Warnings and Precautions that have been approved with estrogen products [50].

8. Calcitonin

Calcitonin is a hormone that decreases osteoclast activity, thereby impeding postmenopausal bone loss. It is indicated for the treatment of women who are more than 5 years post menopause .The intranasal spray is delivered as a single

daily spray that provides 200 IU of the drug. The drug can be delivered subcutaneously, but this route is rarely used. Conflicting evidence exists for fracture reduction with calcitonin and there is insufficient data for its effectiveness in the first few years of menopause. It has analgesic effect after osteoporotic fracture. Side effects include hypocalcemia, nausea, vomiting, allergic reaction and possible increased risk of cancer. Calcitonin is considered second line therapy and no longer widely used for treatment of osteoporosis [51].

9. Conclusion

Osteoporosis represents a significant public health challenge, necessitating a multifaceted approach to reduce fracture incidence and improve patient outcomes. By understanding the pathophysiology, identifying risk factors, employing effective diagnostic tools, and implementing evidence-based management strategies. Management strategies include general measures such as weight-bearing exercise, adequate calcium and vitamin D intake, smoking cessation, and moderation of alcohol consumption. Pharmacotherapy is indicated for patients at significant risk of osteoporosis or fracture. If pharmacologic therapy is indicated, options include bisphosphonates, RANK ligand inhibitor, parathyroid hormone-receptor agonists, estrogen related therapies and sclerostin inhibitor. Decisions must be individualized and should include the patient in the process of shared decision-making.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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