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A Review on comparative efficacy of Alendronate and Risedronate on bone mineral density in Osteoporosis patients

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Abstract

Osteoporosis has emerged as a common skeletal abnormality indicated by reduced bone mineral density (BMD) and structural changes/deterioration of bone tissue. This disorder eventually turns an individual into a fracture-prone one. Among the therapeutic agents currently available in the market, bisphosphonates such as Alendronate and Risedronate occupy a commanding place because they are very effective in the management of bone loss and the gain in BMD. This review article brings together efficacy, safety, and clinical outcomes between Alendronate and Risedronate in osteoporosis management. Alendronate appears to have a stronger affinity for bone binding and a longer retention period in the skeleton; Risedronate, however, offers stronger speed of action and better gastrointestinal tolerability. Both these drugs have shown very effective in improving BMD and decreasing the occurrence of vertebral and non-vertebral fractures. The selection between these drugs should depend on certain factors about the individual patient-that would include fracture risk, co-morbidities, tolerability, and adherence. Future promising directions for osteoporosis therapy include individualized treatment, dependence on bone turnover markers, and novel combinations of therapies to further improve bone strength and long-term outcomes.

Keywords: Osteoporosis, BMD, therapeutic agents, bone strength

Introduction

Osteoporosis is a chronic metabolic bone abnormality indicated by decreased mineral density of bone (BMD) and increased fragility of bone, predisposing the individual to fractures. It is generally called as a "silent disease" as it is asymptomatic due to the slow progression of bone destruction until fractures happen, most seen in the hip, or spine or wrist. Osteoporosis is another world-wide disease affecting millions, especially elderly people. Increased incidences are seen for postmenopausal women who experience decreased estrogen levels that are vital for sustaining bone health. However, men and younger adults, and in some rare cases, children may be affected with some level of osteoporosis depending on their medical condition or lifestyle that may otherwise contribute to bone health.[1,2]

Osteoporosis occurs as a consequence of the dysregulation of formation and resorption of bone thus causing net bone loss. Risk factors may potentiate accelerated loss of bone, including smoking and high alcohol intake, little exercise, dietary deficiency of Ca^{+2} (Calcium) and vitamin D, medications such as corticosteroids, & illnesses like rheumatoid arthritis and some endocrine disorders. Bone loss is typically insidious and painless; hence the majority of individuals do not know about their condition until sustaining a fracture, which can greatly impair their mobility and quality of life, especially among the elderly. The burden that osteoporosis causes on public health and healthcare systems is significant, not only because of the immediate effects of fractures, but also due to the long-term disability, dependence, and increased mortality associated with complications.[3,4]

Types of osteoporosis

Type	Sub-type	Description	Common Causes/Risk Factors	Common Fracture Sites
Primary Osteoporosis	**Type I (Postmeno pausal)**	Seen in women with menopause because of estrogen deficiency	Menopause, hormonal changes	Vertebrae, wrist
	Type II (Senile)	Bone loss related to Age-related factors in both men and women, typically after age 70	Aging, reduced calcium/vitamin D absorption	Hip, pelvis
Secondary Osteoporosis	No sub-types; classified by cause	Caused by underlying diseases or medications affecting bone health	Corticosteroids, endocrine disorders, CKD, RA, alcohol, smoking	Any skeletal site, commonly spine/hip[5,6]

Stages

Stage	Bone Mineral Density Status	T-Score Range
Normal	Normal	≥ -1.0
Osteopenia	Low bone mass	-1.0 and -2.5
Osteoporosis	Significantly reduced	T-score ≤ -2.5
Severe Osteoporosis	Osteoporosis with one or more fractures	≤ -2.5 + fracture

Pathogenisis

Disruption inbalance between bone loss and bone formation mechanism causes osteoporosis, leads to decreased bone mass and structural changes. Bone remodeling is a cyclic process that involves the resorption of old bone by the action of osteoclasts followed by the formation of new bone through osteoblastic action.

In osteoporosis, there is abnormal increase in osteoclasts and abnormal decrease in osteoblastsleads to disruption in balance causing more bone loss. Several factors disturb the balance in these patients: old age, hormonal changes, genetic predisposition, and deficiency of calcium and vitamin D. Chronic inflammation, oxidative stress, and elevated levels of parathyroid hormone (PTH) do play a detrimental role in bone loss.[9,10]

RANK/RANKL/OPG signaling pathway at the molecular level is essential for bone metabolism. Receptor activator of nuclear factor-kappa B ligand (RANKL) binds its receptor RANK on osteoclast precursors, which favors osteoclast differentiation and bone resorption.

Osteoprotegerin (OPG) serves as a decoy receptor for RANKL and inhibits this process. In osteoporosis, reduced levels of OPG, along with increased levels of RANKL expression, lead to excessive activity of osteoclasts and breakdown of bone.[11,12]

Accompanying factors are the raise in inflammatory cytokines that can include TNF-α and IL-6, glucocorticoid-induced bone loss, as well as decreased mechanical loading due to sedentary lifestyles. This pathological mechanism results in fragile bones, such that people are very open to fractures, particularly in the spine, hip, and wrist.[13,14]

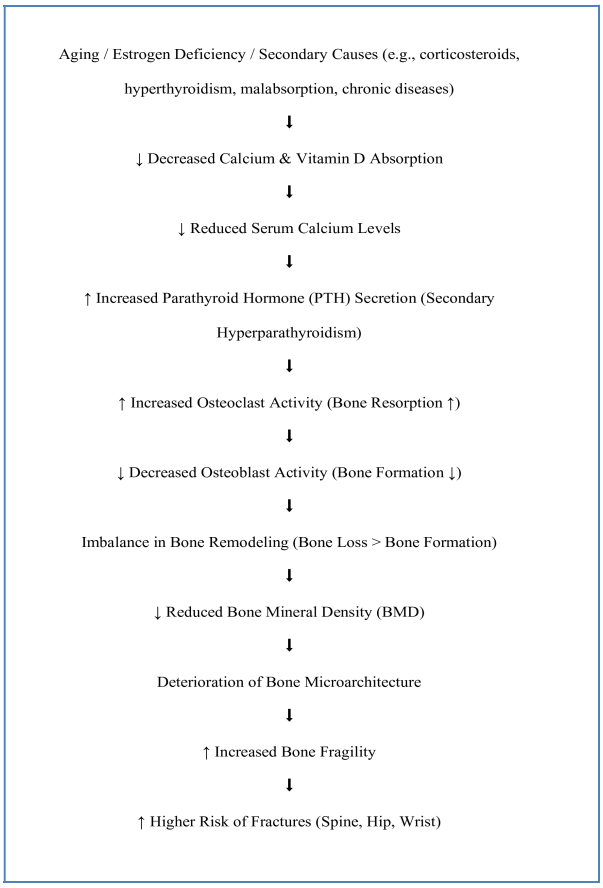

Figure1 : Pathogenesis of osteoporosis

Symptoms

Category	Causes
Primary Osteoporosis	- Postmenopausal (Type 1): Estrogen deficiency increases bone resorption.
	- Age-related (Type 2): Reduced calcium absorption and osteoblast activity with aging.
Secondary Osteoporosis	- Endocrine Disorders: Hyperthyroidism, Cushing's syndrome, diabetes, hypogonadism.
	- Medications: Long-term corticosteroids, anticonvulsants, heparin, aromatase inhibitors.
	- Nutritional Deficiencies: Calcium, vitamin D deficiency, malabsorption (celiac disease).
	- Lifestyle Factors: Smoking, alcohol abuse, physical inactivity, low BMI.[15,16]

Treatment

1. Lifestyle Modifications

Osteoporosis management has a huge bearing on lifestyle. An adequate calcium intake and vitamin D are therefore inevitable since they support bone structural health. Exercise such as walking, jogging, and resistance training improves bone strength and reduces the risk of fractures. Smoking and drink restriction makes sense since both are contributory to bone loss. Fall prevention strategies - modifying houses for safety and balance exercises - help prevent fractures and fractures in elderly patients.[17,18,19]

2. Pharmacological Therapy

Bisphosphonates are first-line treatments that inhibit activity of osteoclast cells and decrease bone resorption. Examples includes Alendronate, Risedronate, Ibandronate, and Zoledronic acid. Denosumab, a monoclonal antibody against RANKL, is given to patients unable to tolerate bisphosphonates. Selective Estrogen Receptor Modulators (SERMs), such as Raloxifene, are helpful for postmenopausal women as they mimic the bone-protective effects of estrogen. Teriparatide and Abaloparatide, which are analogs of parathyroid hormone, stimulate bone formation in cases of very severe osteoporosis.[20]

A. Anti-Resorptive Agents (Inhibit Bone Resorption)

Class	Drug names	Mechanism of Action
Bisphosphonates (1st-Line Therapy)	Alendronate, Risedronate, Ibandronate, Zoledronic Acid	The action of inhibiting osteoclast-mediated resorption of bone is achieved through the binding of hydroxyapatite in bones.
Selective Estrogen Receptor Modulators (SERMs)	Raloxifene, Bazedoxifene	Mimic the effects of estrogen on the bones but do not encourage breast or uterine tissues.
RANKL Inhibitor	Denosumab	Monoclonal antibody which inhibits RANKL thus reducing osteoclast activity and bone loss.
Calcitonin	Salmon Calcitonin (Nasal Spray, Injection)	It inhibits the function of osteoclast in a direct way reducing bone resorption.
Hormone Replacement Therapy (HRT)	Estrogen, Estrogen + Progestin	Prevents postmenopausal bone loss due to maintenance of estrogen levels.[21,22]

B. Anabolic Agents (Stimulate Bone Formation)

Class	Drugs	Mechanism of Action
Parathyroid Hormone (PTH) Analogues	Teriparatide (PTH 1-34), Abaloparatide	Stimulates osteoblasts and improves new bone formation
Sclerostin Inhibitor	Romosozumab	The sclerostin inhibitors increase osteoblast activity; hence, they promote bone formation

C. Adjunctive Therapies

Class	Drugs	Mechanism of Action
Calcium and Vitamin D Supplements	Calcium and Vitamin D	Essential for bone mineralization and calcium absorption.
Strontium Ranelate (Less Commonly Used)	Strontium Ranelate	It increases osteoblast activity and decreases osteoclast activity.[23,24]

3. Hormone Replacement Therapy (HRT)

Estrogene therapy proves quite effective in keeping up the bone density of a postmenopausal woman and therefore diminishes her chances of having fractures . Hormone replacement therapy is, however, very often indicated just for younger postmenopausal women and those identified as very high risk for fractures in the absence of contraindications due to an increased risk of cardiovascular diseases and breast cancer. Calcitonin therapy may also provide some short-term pain relief for vertebral fractures.

4. Surgical Interventions

In case of fractures in severe osteoporosis condition, through surgical interventions, these can aid in the stabilization of vertebral fractures and pain relief through vertebroplasty or kyphoplasty. Patients with fractures that preclude ambulation may require a hip or joint replacement. Usually, surgery is indicated owing to the failures of medical management or if the fractures severely affect quality of life.[25,26,27]

Bone Structure and Bone Mineral Density (BMD)

Bone is a special connective tissue with structural, protective, and metabolic functions, and bone features organic and inorganic materials to provide strength and some flexibility. Basically, bone can be generally classified into two categories: cortical (compact) bone, which is dense and constitutes the outer covering of bone, and trabecular (spongy) bone, which technically is lighter in weight and lies inside bone, principally on the ends of long bones and the great vertebrae. Collagen fibers, in particular, Type I collagen, render the matrix flexible and tensile, while hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$], an inorganic mineral phase, is responsible for giving hardness and rigidity to the matrix. [28,29]

Minerals are indeed very important in bone integrity and in metabolic functions. Calcium is the main component mineral in bone. About 99% of the body's total calcium stores are found in the skeleton. It ionically bonds with phosphate to form hydroxyapatite crystals that become embedded in a fibrous collage matrix and confer compressive strength to bone. In addition to calcium and phosphate, several other minerals,

including magnesium, fluoride, sodium, and zinc, are also necessary for the catabolic enzymatic and structural stability of the bone. Metabolism of calcium and phosphate is under very tight control by hormones, such as parathyroid hormone (PTH), calcitriol vitamin D, and calcitonin, ensuring equilibrium between bone resorption and formation. The deficiency of these minerals, especially calcium and vitamin D, brings disorders of the bones like osteoporosis, osteomalacia, and poor healing of the bones. Therefore, healthy mineral intake and hormonal balance are crucial for bone health throughout an individual's life.

Bone Mineral Density (BMD) can be defined as the total analysis of the concentration of minerals, particularly calcium as well as phosphorus, found in the bone tissue, thus one of the prime indicators of bone strength as well as overall skeletal health.

Bones are the main stores of calcium for the body, which has to be adequately mineralized to provide structural integrity. Currently, the measurement of BMD is mainly done through a Dual-Energy X-ray Absorptiometry (DEXA or DXA) scan, which has become a gold standard. Indeed, this testing is pain-free, non-invasive, and provides quantitative data in mineral density at predetermined anatomical sites such as the lumbar spine, hip, and femoral neck, where the risk of osteoporotic fractures is highest. There are some other non-DXA techniques used to derive bone density results like quantitative computed tomography (QCT) and ultrasound, but the predominant choice remains DEXA, thanks to precision and low-risk radiation hazards.[30,31]. The results of the BMD measurement are given in both a T-score and a Z-score. The T-score compares the individual's BMD with that of a healthy young adult of the same sex, while the Z-score compares it against norms matched for age and sex.[32,33]

T-Score Range	Bone Health Status	Interpretation
≥ -1.0	Normal	Bone density is considered normal.
Between -1.0 to -2.5	Osteopenia (Low bone mass)	Bone density is lower than normal but not low enough to be classified as osteoporosis.
≤ -2.5	Osteoporosis	Bone density is low enough to be classified as osteoporosis.
≤ -2.5 with fractures	Severe (Established) Osteoporosis	Osteoporosis accompanied by one or more fragility fractures.

Figure 1 :Bone Mineral Density (BMD) Assessment and T-Score Interpretation

Role of bisphosphonates in the treatment of osteoporosis

Among all the pharmacotherapy provided, the bisphosphonates have the first-line treatment for being the most efficacious agents to reduce bone resorption, enhance bone mineral density (BMD), and prevent fractures. They inhibit the breakdown of bones by osteoclasts, which will normalize the proportion between the formation and destruction of bone tissues. The effects of therapeutic success, Alendronate, and Risedronate format have contributed to the overall success of osteoporosis management and are now primary agents in long-term maintenance of bone health.

1. Inhibition of Osteoclast Activity

The main action of bisphosphonates is on inhibition of osteoclasts, the cells that carry out bone destruction activity. Bisphosphonates have a strong affinity for hydroxyapatite, the mineral part of the bone, and thus they get lodged in the bone matrix. When osteoclasts resorb the bone, they also ingest the bisphosphonates that impair their function and lead to osteoclast apoptosis (programmed cell death). This diminishes the already ongoing degradation processes of bone and thus limits further loss, and this is an important mechanism in the treatment of osteoporosis.

2. Preservation of Bone Mass

Imbibition of osteoclast activity (Bone destruction mechanism), thus decreasing the bone destruction rate maintaining equal proportion between bone resorption and formation. This promotes bone production by osteoblasts without further obstructionfrom bone destruction process by osteoclasts. At the end, this leads to a gain or stabilization of bone mass, particularly in the spine and hip where osteoporotic fractures are most common.[34,35]

3.Improvement in Bone Mineral Density (BMD)

By inhibiting bone resorption while allowing for continued bone formation, bisphosphonates help increase or stabilize BMD. Clinical studies have documented significant improvements in BMD at important skeletal sites like the lumbar spine, femoral neck, and total hip. High BMD translates into low bone fragility and low fracture risk, one target of therapy for patients suffering from osteoporosis.

4.Reduction in Fracture Risk

The most clinically important outcome of bisphosphonate therapy is the decrease in fracture incidence. Evidence from numerous studies has demonstrated that bisphosphonates can reduce the risk of vertebral fractures by up to 70%; hip fractures by 40-50%; and non-vertebral fractures by around 30%. Therefore, it is highly effective in preventing disabilities and thus improving quality of life among osteoporosis patients, especially in postmenopausal women and older patients.[36,37]

Alendronate: Efficacy and safety in osteoporosis

1. Efficacy of Alendronate

Alendronate is a bisphosphonate widely used as a first-line treatment for the prevention and treatment of osteoporosis. It prevents bone destruction andimproves bone mineral density through inhibition of osteoclast activity. From large clinical trials including the Fracture Intervention Trial (FIT), it has been demonstrated that alendronate given daily or weekly decreases the risk of vertebral, hip and wrist fracture. Any improvement in BMD is noticed within 6 months to 12 months after initiation of therapy, whereas maximal effects are reached usually after 2–3 years of continuous therapy. Alendronate also helps maintain bone mass in patients with glucocorticoid-induced osteoporosis and in men with osteoporosis.[38]

2. Safety Profile of Alendronate

Alendronate is often very well tolerated, especially when taken orally (usually 70 mg once weekly). However, it can produce certain side effects, notably gastrointestinal (GI) effects such as esophagitis and gastritis, but also generalized abdominal pain. To help prevent these effects, patients should be advised to take medication with proper water and remain straight for at least 30 minutes after administration. The more severe side effects, which are less common, include osteonecrosis of the jaw (ONJ), especially in patients with cancer treated with high doses intravenously, and atypical femoral fractures with long-term use. The probabilities of such events are generally quite low and are more frequently outweighed by the benefits in patients at increased risk of fractures.

3. Monitoring and Considerations

A DEXA scan for bone mineral density is routinely done every 1-2 years to assess treatment response. It is advisable to check a patient's serum calcium and vitamin D levels before commencing therapy. Alendronate is contraindicated in the following cases: patients with esophageal abnormalities, inability to sit upright for 30 minutes, hypocalcemia, and severe renal impairment (creatinine clearance <35 mL/min). Adequate calcium and vitamin D intake during treatment is essential for maximal benefit. A treatment will be greatly affected if the patient does not comply with administration; missed doses or further improper administration can serve to greatly diminish efficacy.[39,40]

Risedronate: Efficacy and safety in osteoporosis

1. Efficacy of Risedronate

Risedronate is a 3rd generation bisphosphonate that effectively reduces bone destruction bysupressing osteoclast activity and leading to apoptosis of these bone-resorbing cells. It has shown high effectiveness inimproving bone mineral density (BMD) and reducing fracture risk in various populations, especially in postmenopausal women and patients on long-term corticosteroids. Clinical trials, such as the VERT (Vertebral Efficacy with Risedronate Therapy) studies, have demonstrated that risedronate significantly reduces vertebral fractures by up to 41–49% and non-vertebral fractures by 30–40% within 1–3 years of therapy. It begins to show effects within 6 months and is comparable in efficacy to alendronate.

2. Safety Profile of Risedronate

Generally well tolerated when given per guidelines, risedronate's common side effects affect the gastrointestinal system: dyspepsia, abdominal pain, and nausea. It may be better tolerated from a gastrointestinal perspective than alendronate. Rarely, adverse events of interest include osteonecrosis of the jaw (ONJ) and atypical femoral fractures on prolonged use (>5 years). The likelihood of hypocalcemia is low, yet it may still arise, particularly in individuals with vitamin D deficiency. Safety can be improved with proper administration-seeing that the patient takes the tablet with water while remaining upright for 30 minutes.[41,42]

3. Monitoring and Clinical Considerations

Therapy should not commence before serum calcium and vitamin D level assessment and correction if found deficient. BMD testing via DEXA is done at both baseline and periodically (every 1-2 years) to monitor responsiveness. Renal function has to be considered-risedronate is contraindicated in patients with severe renal impairment (creatinine clearance <30 mL/min). Adequate hydration, taking the drug in an upright position with proper timing (in fasting states), helps minimize GI side effects and maximize absorption. After 3-5 years of treatment, based on fracture risk assessment, a "drug holiday" is contemplated.[43]

Comparitive analysis of alendronate and risedronate

1.Mechanism of Action

Feature	Alendronate	Risedronate
Drug Class	Nitrogen-containing bisphosphonate	Nitrogen-containing bisphosphonate
Target	Inhibits farnesyl pyrophosphate synthase (FPPS) in osteoclasts	Same
Bone Affinity	High	Moderate
Effect	Strong inhibition of bone resorption	Rapid inhibition of bone resorption
Duration of Action	Long-lasting	Shorter skeletal retention[44]

2.Pharmacokinetics

Parameter	Alendronate	Risedronate
Oral Bioavailability	~0.6%	~0.63%
Bone Binding Affinity	High	Moderate
Skeletal Half-Life	Up to 10 years	Around 2 years
Peak Action	6–12 months	3–6 months
Excretion	Unchanged in urine	Unchanged in urine[45]

3.Clinical Efficacy

Parameter	Alendronate	Risedronate
Lumbar Spine BMD Increase	5–8% over 3 years	4–6% over 3 years
Femoral Neck BMD Increase	3–6%	2–4%
Vertebral Fracture Risk Reduction	~50%	~41%
Hip Fracture Risk Reduction	~40%	~30%
Non-Vertebral Fracture Risk	Moderate reduction	Moderate reduction
Time to Observe Clinical Effect	6–12 months	3–6 months[46]

4. Tolerability and Safety

Side Effect	Alendronate	Risedronate
GI Upset (nausea, heartburn)	Common	Less frequent
Esophageal Irritation	More common	Rare
Musculoskeletal Pain	Possible	Possible
Osteonecrosis of the Jaw	Rare (long-term use)	Rare (long-term use)
Atypical Femoral Fractures	Rare	Rare
Adherence Issues	Due to GI side effects	Better adherence due to tolerability

5. Dosage and Administration

Parameter	Alendronate	Risedronate
Daily Dose	10 mg	5 mg
Weekly Dose	70 mg	35 mg
Monthly Dose	Not typically used	150 mg
Administration Guidelines	Take with water, upright position for 30 mins, empty stomach	Same
Food Interaction[47,48]	Affects absorption significantly	Same

Future perspectives:

The relative effectiveness of Alendronate and Risedronate in enhancing bone mineral density (BMD) in patients suffering from osteoporosis is of continued clinical interest that should inform future practice. Both agents have significantly improved BMD and reduced bone fracture risk; however, future studies must consider longer-term outcome measures, safety profiles, and quality of life with extended administration. Drug therapy could possibly be more individualized by utilizing genetic markers, bone turnover markers, and patient-specific risk factors in personalized medicine.

Putting the findings into the real world with bigger and more diverse populations is essential to understand how such drugs perform outside the boundaries of controlled trial mechanisms. Future studies should also look into options of combination therapies, for instance with sequential delivery of anabolic agents and bisphosphonates, to substantiate the general strength and durability of the bone. Research into new formulations for the delivery of these agents, like gastro-resistant tablets or injectable bisphosphonates, would go a long way in improving adherence and tolerability. On the whole, the convergence of clinical, molecular, and technological advancements can enable improved osteoporosis management strategies, which are more effective and personalized.[49,50]

Conclusion

Osteoporosis is a progressive metabolic dysfunction of bone with advancement of bone catabolism characterized by reduction in bone mass and deterioration of bone microarchitecture, with the eventual result of increased fragility and fracture risk. This condition mainly strikes postmenopausal women and the elderly, thus constituting a major public health burden worldwide. Bone mineral density (BMD) is one of the most important parameters to detect osteoporosis and to monitor treatment response; most commonly it is measured at the lumbar spine and hip.

The difference between Alendronate and Risedronate in terms of effectiveness for BMD improvement is dealt with in this review. Both act by inhibiting the osteoclastic bone resorption process and thereby exert their effects on bone strength. Alendronate is characterized by a higher affinity to bone binding and produces a more sustained improvement in BMD, while Risedronate has a faster and better tolerated overall profile, especially regarding the GI side. Individualization of therapy, on the basis of characteristics, risk profiles, and preferences of patients, should be done. In the next few years, personalization of therapy, long-term safety assessment, and the introduction of new treatment options will be the imperatives for osteoporosis management and for improving patient life quality.

References

1. Chin KY, Ng BN, Rostam MKI, Muhammad Fadzil NFD, Raman V, Mohamed Yunus F, Syed Hashim SA, Ekeuku SO. A Mini Review on Osteoporosis: From Biology to Pharmacological Management of Bone Loss. J Clin Med. 2022 Oct 30;11(21):6434
2. Berger C., Goltzman D., Langsetmo L., Joseph L., Jackson S., Kreiger N., Tenenhouse A., Davison K.S., Josse R.G., Prior J.C., et al. Peak bone mass from longitudinal data: Implications for the prevalence, pathophysiology, and diagnosis of osteoporosis. J. Bone Miner. Res. 2010;25:1948–1957.
3. Specker B.L., Wey H.E., Smith E.P. Rates of bone loss in young adult males. Int. J. Clin. Rheumtol. 2010;5:215–228.
4. Mitchell P.J., Chan D.D., Lee J.K., Tabu I., Alpuerto B.B. The global burden of fragility fractures—what are the differences, and where are the gaps. Best Pract. Res. Clin. Rheumatol. 2022:101777.
5. Dobbs MB, Buckwalter J, Saltzman C. Osteoporosis: the increasing role of the orthopaedist. Iowa Orthop J. 1999;19:43-52.
6. Ji MX, Yu Q. Primary osteoporosis in postmenopausal women. Chronic Dis Transl Med. 2015 Mar 21;1(1):9-13.

7. Cooper, A., and Cooper, B.B. 1822. A treatise on dislocations, and on fractures of the joints. Churchill. London, United Kingdom. 425 pp.
8. Schapira, D., and Schapira, C. 1992. Osteoporosis: the evolution of a scientific term. Osteoporos. Int. 2:164–167.
9. Albright, F., Bloomberg, E., and Smith, P.H. 1940. Postmenopausal osteoporosis. Trans. Assoc. Am. Physicians. 55:298–305.
10. Riggs, B.L., et al. 1982. Changes in bone mineral density of the proximal femur and spine with aging. Differences between the postmenopausal and senile osteoporosis syndromes. J. Clin. Invest. 70:716–723
11. Prideaux M., Findlay D.M., Atkins G.J. Osteocytes: The master cells in bone remodelling. Curr. Opin. Pharmacol. 2016;28:24–30.
12. Xiao W., Wang Y., Pacios S., Li S., Graves D.T. Cellular and Molecular Aspects of Bone Remodeling. Front. Oral Biol. 2016;18:9–16
13. Ma Q., Liang M., Wu Y., Luo F., Ma Z., Dong S., Xu J., Dou C. Osteoclast-derived apoptotic bodies couple bone resorption and formation in bone remodeling. Bone Res. 2021;9:5.
14. Raisz LG. Pathogenesis of osteoporosis: concepts, conflicts, and prospects. J Clin Invest. 2005 Dec;115(12):3318-25.
15. Riggs BL, et al. Changes in bone mineral density of the proximal femur and spine with aging. Differences between the postmenopausal and senile osteoporosis syndromes. J. Clin. Invest. 1982;70:716–723.
16. Liu YZ, Liu YJ, Recker RR, Deng HW. Molecular studies of identification of genes for osteoporosis: the 2002 update. J. Endocrinol. 2003;177:147–196.
17. Parfitt AM, Villanueva AR, Foldes J, Rao DS. Relations between histologic indices of bone formation: implications for the pathogenesis of spinal osteoporosis. J. Bone Miner. Res. 1995;10:466–473.
18. Aibar-Almazán A, Voltes-Martínez A, Castellote-Caballero Y, Afanador-Restrepo DF, Carcelén-Fraile MDC, López-Ruiz E. Current Status of the Diagnosis and Management of Osteoporosis. Int J Mol Sci. 2022 Aug 21;23(16):9465.
19. Nuti R., Brandi M.L., Checchia G., Di Munno O., Dominguez L., Falaschi P., Fiore C.E., Iolascon G., Maggi S., Michieli R., et al. Guidelines for the management of osteoporosis and fragility fractures. Intern. Emerg. Med. 2019;14:85–102.
20. Cosman F., de Beur S.J., LeBoff M.S., Lewiecki E.M., Tanner B., Randall S., Lindsay R. Clinician's Guide to Prevention and Treatment of Osteoporosis. Osteoporos. Int. 2014;25:2359–2381.
21. Tu KN, Lie JD, Wan CKV, Cameron M, Austel AG, Nguyen JK, Van K, Hyun D. Osteoporosis: A Review of Treatment Options. P T. 2018 Feb;43(2):92-104
22. Cosman F, de Beur S, LeBoff M, et al. Clinician's guide to prevention and treatment of osteoporosis. Osteoporos Int. 2014;25(10):2359–2381.
23. Camacho PM, Petak SM, Binkley N, et al. American Association of Clinical Endocrinologists and American College of Endocrinology: clinical practice guidelines for the diagnosis and treatment of postmenopausal osteoporosis—2016. EndocrPract. 2016;22(suppl 4):S1–S42.
24. Gamsjaeger S, Eriksen EF, Paschalis EP. Effect of hormone replacement therapy on bone formation quality and mineralization regulation mechanisms in early postmenopausal women. Bone Rep. 2021 Mar 23;14:101055.
25. Marcucci G, Brandi ML. Kyphoplasty and vertebroplasty in the management of osteoporosis with subsequent vertebral compression fractures. Clin Cases Miner Bone Metab. 2010 Jan;7(1):51-60.
26. Stevenson J; medical advisory council of the British Menopause Society. Prevention and treatment of osteoporosis in women. Post Reprod Health. 2023 Mar;29(1):11-14.
27. Bandeira L, Lewiecki EM. Anabolic therapy for osteoporosis: update on efficacy and safety. Arch EndocrinolMetab. 2022 Nov 11;66(5):707-716.
28. Šromová V, Sobola D, Kaspar P. A Brief Review of Bone Cell Function and Importance. Cells. 2023 Nov 5;12(21):2576.

29. Weatherholt AM, Fuchs RK, Warden SJ. Specialized connective tissue: bone, the structural framework of the upper extremity. J Hand Ther. 2012 Apr-Jun;25(2):123-31; quiz 132.
30. Florencio-Silva R, Sasso GR, Sasso-Cerri E, Simões MJ, Cerri PS. Biology of Bone Tissue: Structure, Function, and Factors That Influence Bone Cells. Biomed Res Int. 2015;2015:421746.
31. Haseltine KN, Chukir T, Smith PJ, Jacob JT, Bilezikian JP, Farooki A. Bone Mineral Density: Clinical Relevance and Quantitative Assessment. J Nucl Med. 2021 Apr;62(4):446-454.
32. Zhu J, March L. Treating osteoporosis: risks and management. AustPrescr. 2022 Oct;45(5):150-157.
33. Rondanelli M, Gasparri C, Perdoni F, Riva A, Petrangolini G, Peroni G, Faliva MA, Naso M, Perna S. Bone Mineral Density Reference Values in 18- to 95-Year-Old Population in Lombardy Region, Italy. Am J Mens Health. 2022 Sep-Oct;16(5):15579883221119363.
34. Drake MT, Clarke BL, Khosla S. Bisphosphonates: mechanism of action and role in clinical practice. Mayo Clin Proc. 2008 Sep;83(9):1032-45.
35. Kim, B., Cho, Y. J., Lim, W."Osteoporosis therapies and their mechanisms of action (Review)". Experimental and Therapeutic Medicine.2021; 22(6): 1379.
36. Carano A, Teitelbaum SL, Konsek JD, Schlesinger PH, Blair HC. Bisphosphonates directly inhibit the bone resorption activity of isolated avian osteoclasts in vitro. J Clin Invest. 1990 Feb;85(2):456-61.
37. Kim S, Chung YS, Lee Y. Adherence of bisphosphonate and decreased risk of clinical vertebral fracture in osteoporotic patients: A propensity score matching analysis. OsteoporosSarcopenia. 2022 Sep;8(3):98-105.
38. Sharpe M, Noble S, Spencer CM. Alendronate: an update of its use in osteoporosis. Drugs. 2001;61(7):999-1039.
39. Drake MT, Clarke BL, Khosla S. Bisphosphonates: mechanism of action and role in clinical practice. Mayo Clin Proc. 2008 Sep;83(9):1032-45.
40. Emkey R. Alendronate and risedronate for the treatment of postmenopausal osteoporosis: clinical profiles of the once-weekly and once-daily dosing formulations. MedGenMed. 2004 Jul 19;6(3):6.
41. Iwamoto J, Takeda T, Sato Y. Efficacy and safety of alendronate and risedronate for postmenopausal osteoporosis. Curr Med Res Opin. 2006 May;22(5):919-28.
42. Sorensen OH, Crawford GM, Mulder H, Hosking DJ, Gennari C, Mellstrom D, Pack S, Wenderoth D, Cooper C, Reginster JY. Long-term efficacy of risedronate: a 5-year placebo-controlled clinical experience. Bone. 2003 Feb;32(2):120-6.
43. Liberman UA, et al. Effect of alendronate on bone mineral density and the incidence of fractures in postmenopausal osteoporosis. N Engl J Med.1995. 333:1437–1443
44. Levis S, Quandt et al Alendronate reduces the risk of multiple symptomatic fractures: results from the fracture intervention trial. J Am Geratr Soc 50:409–415
45. Emkey R. Alendronate and risedronate for the treatment of postmenopausal osteoporosis: clinical profiles of the once-weekly and once-daily dosing formulations. MedGenMed. 2004 Jul 19;6(3):6.
46. Christiansen, C. & Phipps, Roger &Burgio, D. & Sun, L. & Russell, Dylan & Keck, Bradly. Comparison of risedronate and alendronate pharmacokinetics at clinical doses. Osteoporos Int.2003; 14 : S38.
47. Cadarette SM, Katz JN, Brookhart MA, Stürmer T, Stedman MR, Levin R, Solomon DH. Comparative gastrointestinal safety of weekly oral bisphosphonates. Osteoporos Int. 2009 Oct;20(10):1735-47.
48. Kennel KA, Drake MT. Adverse effects of bisphosphonates: implications for osteoporosis management. Mayo Clin Proc. 2009 Jul;84(7):632-7; quiz 638

49. Lippuner K. The future of osteoporosis treatment – a research update. Swiss Med Wkly. 2012;142(2930):w13624.
50. Lorentzon M. Treating osteoporosis to prevent fractures – current concepts and future developments (Review). J Intern Med 2019; 285: 381–394.

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