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Review Article

Evidence-based expert recommendations for the diagnosis and targeted management of osteoporosis

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Abstract

To derive practical, experience-driven clinical insights related to evidence-based statements for diagnosing and managing osteoporosis. The meeting involved in-depth discussions and a review of recent literature and real-world clinical perspectives. A set of 32 evidence-based statements was developed following a literature search of English articles from 2018 to 2023, using keywords such as 'Osteoporosis', 'Diagnosis', 'Romosozumab' and 'anti-osteoporosis'. Two virtual national meetings were held with 23 experts in Orthopaedics, Rheumatology and Endocrinology. According to the evidence-based recommendation, osteoporosis frequently progresses as a "silent disease," showing no signs until a fracture happens. The risk varies by age, gender and other factors. Screening is recommended for postmenopausal women over 50 years and men over 60 years of age due to their higher risk of bone loss. The dual-energy X-ray absorptiometry scan is the gold standard for diagnosing osteoporosis. It should be repeated every 1-2 years to track bone mineral density (BMD). The Fracture Risk Assessment Tool helps to evaluate the risk of fragility fractures. Bone turnover markers help assess the effectiveness of treatment. Anti-osteoporosis treatments are recommended for those at a high or very high fracture risk. Anti-resorptive therapies are suitable for high-risk patients, whereas anabolic therapies are preferred for very high-risk cases. Romosozumab, a dual-action medication, is effective in postmenopausal women and has been approved for men in Japan and Australia as of December 2024. The ideal candidates for Romosozumab include those with declining BMD, despite anti-resorptive treatment or a high fracture risk.

Keywords: Osteoporosis, Postmenopausal, Dual-energy X-ray absorptiometry, Fracture risk assessment tool, Anabolics, Anti-resorptive, Romosozumab, Denosumab.

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1. Introduction

The public health system has a significant challenge from osteoporosis, which affects patients' quality of life by having social and psychological effects.[1,2] Reduced bone mass and degeneration of bone architecture are the hallmarks of this progressive systemic bone illness, which increases bone fragility and fracture risk.[2,3] Osteoporosis-related fractures can cause significant pain, suffering and disability.[4] 1 in 3 females aged >50 years and 1in 5 males will experience an osteoporotic fracture in their lifetime.[5] Osteoporosis develops when bone resorption outpaces bone formation, with increased osteoclast activity causing more bone breakdown (**Figure 1**).[6]

1.1. Prevalence of osteoporosis

Globally, almost 200 million people are affected with osteoporosis. Every 3 seconds, there is a fragility fracture happening somewhere in the world. India has one of the

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greatest burdens, with one in eight males and one in three females being affected. Globally, hip fracture rates are expected to rise by 310% in men and 240% in women by 2050.[7-9]

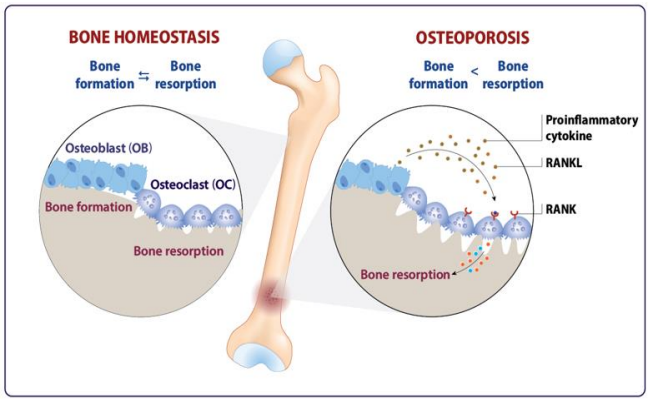

Figure 1: Increased osteoclast activity leading to net bone loss in osteoporosis[6]

1.2. Aetiology

Osteoporosis can occur either due to ageing or due to some comorbid conditions (**Table 1**).[10]

1.3. Diagnosis of osteoporosis in postmenopausal women (AACE 2020)[11]

According to the American Association of Clinical Endocrinologists (AACE) 2020, the diagnostic criteria for osteoporosis in postmenopausal females are:

1. T-score of -2.5 or below in the lumbar spine, femoral neck, total proximal femur or 1/3 radius
2. Low-trauma spine or hip fracture (*regardless of BMD*)
3. T-score between -1.0 and -2.5 and fragility fracture of the proximal humerus, pelvis or distal forearm
4. T-score between -1.0 and -2.5 and high fracture risk assessment tool (FRAX®) (or if available, trabecular bone score [TBS]-adjusted FRAX®) fracture probability based on country-specific thresholds.

1.4. Osteoporosis treatment

By regulating bone growth and resorption, osteoporosis treatment aims to strengthen bones and reduce fracture risk. The two primary types of drugs used to treat osteoporosis are: Anabolic (forming bone); anti-resorptive (slowing resorption).

1.5. Clinically used anti-osteoporosis drugs

Pharmacological therapies utilized for the management of osteoporosis are mentioned in **Table 2**.[12-16]

1.6. Romosozumab

Romosozumab is the Food and Drug Administration (FDA)-approved monoclonal immunoglobulin G2 antibody that targets the Wnt antagonist Sclerostin (secreted by osteoblasts). Romosozumab has been approved by the Central Drugs Standard Control Organisation to treat osteoporosis in postmenopausal females who are at high risk of fractures, such as those who have a history of osteoporotic fractures, have several risk factors for fractures, or have not responded to or are unable to tolerate other osteoporosis treatments.[17,18]

The methodology followed to derive practical, experience-driven clinical insights related to evidence-based statements for diagnosing and managing osteoporosis is mentioned below:

We devised a set of 32 evidence-based statements by performing a detailed literature search. This search included English articles published from 2018 to 2023 and involved keywords such as ‘Osteoporosis’, ‘Diagnosis’, ‘Romosozumab’ and ‘Anti-osteoporosis’. Studies involving animal models and articles in languages other than English were excluded. For each statement, we conducted a rigorous independent literature search to obtain top-tier evidence. Two virtual national meetings were convened, involving 23 renowned experts from the fields of Orthopaedics, Rheumatology and Endocrinology.

The primary objectives of this expert panel deliberation were to derive practical, experience-based clinical insights pertaining to the recommended statements. The meeting included in-depth discussions, exploring recent literature and real-world clinical insights related to these statements.

Table 1: Aetiology of primary and secondary osteoporosis[10]

Type of osteoporosis	Aetiology
Primary osteoporosis	Linked to ageing and deficiencies in sex hormones such as oestrogen, especially in postmenopausal women, and in men, increased levels of sex hormone-binding globulin inactivate testosterone and oestrogen
Secondary osteoporosis	Caused by various comorbid conditions such as Cushing's syndrome, rheumatoid arthritis and/or medications such as glucocorticoids

Table 2: Clinically used anti-osteoporosis drugs[12-16]

Drug	Route and dosing	Potential adverse effects	Contraindications	Other considerations
Anti-resorptive agents				
Bisphosphonates				
Alendronate	Oral: 70 mg weekly *or* 10 mg daily	• Oesophageal or GI intolerance • MSK discomfort Rare: AFF, ONJ	• CrCl < 30–35 mL/min • Oesophageal abnormalities • Inability to be upright > 30 min • Hypocalcaemia	• Foods, drinks (except plain water), other drugs should be avoided for > 30–60 min • Minerals and dairy impair absorption if taken close together
Risedronate	Oral: 35 mg weekly or 150 mg monthly or 5 mg daily			
Zoledronic acid	Intravenous: 5 mg yearly	• Transient flu-like symptoms • Hypocalcaemia • Renal toxicity • Rare: AFF, ONJ	• CrCl < 35 mL/min • Hypocalcaemia	• Inadequate vitamin D increases risk for hypocalcaemia • Less frequent dosing than yearly may be considered
Ibandronate	150 mg once every month	Hypocalcaemia		• Not very effective in hip and non-vertebral fracture prevention
RANK-Ligand inhibitor (monoclonal antibody)				
Denosumab	Subcutaneous: 60 mg every 6 months	• Hypocalcaemia • Dermatitis, infections • MSK discomfort • Rare: AFF, ONJ	• Hypocalcaemia	• Inadequate vitamin D increases risk for hypocalcaemia • Caution warranted in severe renal impairment • Rapid bone loss and risk of vertebral fractures if delayed dose or with discontinuation. It is safe for renal patients with eGFR ≤ 30 mL/min/1.73 m^2 caution for hypocalcaemia in advanced kidney disease
Hormonal therapy				
Menopausal hormonal therapy	Multiple regimens	• VTE, CVD and stroke • Breast cancer	• VTE, CVD, stroke, oestrogen-dependent tumours, abnormal vaginal bleeding, and active liver disease	• Only in postmenopausal women
Raloxifene (SERM)	Oral: 60 mg daily	• VTE, CVD and stroke • Vasomotor symptoms and leg cramps	• VTE, CVD, stroke and abnormal vaginal bleeding	
Anabolic agents				
Parathyroid hormone analogue				
Teriparatide	Subcutaneous: 20 µg daily for 24 months	• Orthostatic hypotension, nausea • Hypercalcaemia, hypercalciuria • MSK discomfort	• CrCl < 30 mL/min • Bone malignancy, Paget disease, previous skeletal radiation • Hypercalcaemia disorder • Unexplained elevated ALP	• Caution warranted with active or previous kidney stone disease
Sclerostin inhibitor				
Romosozumab	210 mg subcutaneous monthly for 12 months	• Arthralgia, headache • Rare: ONJ, AFF	• Hypocalcemia	• Precaution: Not to use if myocardial infarction or stroke within preceding one year

Abbreviations: AFF: Atypical femoral fracture; ALP: Alkaline phosphatase; CrCl: Creatinine clearance; CVD: Cardiovascular disease; GI: Gastrointestinal, MSK: Musculoskeletal; ONJ: Osteonecrosis of the jaw; RANK: Receptor activator of nuclear factor κ-β; SERM: Selective oestrogen receptor modulator; VTE: Venous thromboembolism

2. Discussion

The following are the final expert recommendations and their supporting evidence available in the literature.

2.1. Prevalence

2.1.1. Expert recommendation

Across different age groups of Indian women, the prevalence of osteoporosis ranges from 8% to 62%. Likewise, osteoporosis prevalence in males aged >50 years ranges from 8.5% to 24.6%, but it may differ as per age group stratification.

2.1.1.1. Evidence

India, with a population of over 1.3 billion, has around 230 million people aged ≥50 years. Osteoporosis affects about 20% of females over 50 years of age, with prevalence ranging from 8% to 62% in other age categories.[19] Globally, osteoporosis affects 10% of women in their 60s, 20% in their 70s, 40% in their 80s and 66% in their 90s.[20] In Indian men aged >50 years, prevalence varies from 8.5% to 24.6%.[19]

2.2. Osteoporosis

2.2.1. Expert recommendation

Osteoporosis is a silent disease and a chronic condition, similar to diabetes and hypertension. It needs early diagnosis, surveillance, vigilance and lifelong management.

2.2.1.1. Evidence

Osteoporosis is a chronic, incurable condition such as heart disease, diabetes or hypertension, which requires lifelong management and significantly impacts both individuals and the healthcare system.[21,22] Osteoporosis is a 'silent' disease, frequently overlooked until a fracture occurs. It primarily affects postmenopausal women and older men, with fractures more often in the hip, spine and wrist.[23] Prevention of osteoporosis is possible through early diagnosis, before a fracture occurs, by evaluating BMD and initiating timely treatment.[5]

2.3. Screening

2.3.1. Expert recommendation

Osteoporosis screening should be done for postmenopausal females >50 years and males >60 years of age.

2.3.1.1. Evidence

The Indian Society for Bone and Mineral Research (ISBMR) recommends screening for osteoporosis in females aged ≥60 years as well as in males aged ≥65 years. Postmenopausal females aged <60 years and males aged between 50 and 64 years should be assessed for osteoporosis, if their clinical risk factors indicate potential concerns.[19]

2.3.2. Expert recommendation

Osteoporosis screening should be done in the following patients:

1. Patients with a high risk for bone loss or fractures and those suffering from diseases such as rheumatological diseases, endocrinological diseases or gastrointestinal (GI) diseases, which can cause secondary osteoporosis
2. Patients taking certain medications for a long term, such as steroids or anti-epileptics
3. Young patients with fragility fractures
4. Patients who are chronic smokers or have testosterone deficiency.

2.3.2.1. Evidence

Osteoporosis screening to be performed in patients with the following risk factors:[5,24-28]

1. Secondary causes include rheumatoid arthritis, GI diseases, endocrine disorders such as adrenal insufficiency, Cushing's syndrome and diabetes mellitus
2. Patients taking steroids and antiepileptic drugs
3. Young adults with fragility fractures and chronic smokers
4. Testosterone deficiency syndrome

2.4. Diagnosis and monitoring

2.4.1. Expert recommendation

Before initiating any osteoporosis therapy, it is essential to assess and monitor serum calcium, serum creatinine, parathyroid hormone (PTH), vitamin D, serum phosphorus and alkaline phosphatase (ALP) levels.

2.4.1.1. Evidence

The literature suggests that before initiating any treatment, all patients with osteoporosis should be assessed for potential secondary causes. The following tests are recommended to rule out secondary osteoporosis:[5]

1. Complete blood count
2. Serum levels of creatinine, calcium, phosphorus and magnesium
3. Liver function tests, including alkaline phosphatase
4. Vitamin D (25[OH]D)
5. Parathyroid hormone (PTH)
6. Total testosterone and gonadotropin levels in younger men

2.4.2. Expert recommendation

When diagnosing osteoporosis, a dual-energy X-ray absorptiometry (DEXA) scan is the gold standard. However, if a DEXA scan is not available, then a good quality lateral thoracolumbar spine X-ray could be used as a screening tool to rule in or rule out osteoporotic vertebral fractures.

2.4.2.1. Evidence

The current gold standard for diagnosing osteoporosis, monitoring its progression and evaluating treatment effectiveness is DEXA.[29] Besides, lateral lumbar spine radiographs, with or without anteroposterior views, are used to detect vertebral compression fractures.[30]

2.4.3. Expert recommendation

DEXA to be repeated at least 1–2 years in order to check improvement in BMD. However, the DEXA scan has limitations, including the need to use the same machine consistently, as variations in settings and configurations can occur with different machines. Additionally, the accessibility of DEXA scanning is limited.

2.4.3.1. Evidence

The National Osteoporosis Foundation (NOF) recommends repeating BMD tests 1–2 years after initiating treatment and every 2 years thereafter, with more frequent testing in certain cases. Similarly, the American Association of Clinical Endocrinologists (AACE) recommends that DEXA scans of the lumbar spine and hips be performed every 2 years.[31]

2.4.4. Expert recommendation

The FRAX® tool is a valuable clinical tool for assessing the risk of fragility fractures in individuals at risk of osteoporosis.

2.4.4.1. Evidence

It is now recommended that fracture probability should be considered when making treatment decisions. Thus, BMD results, along with clinical risk factors should be considered, e.g., FRAX® tool for accurate assessment of the fracture risk and provide treatment decisions. FRAX®, developed by the WHO, is a global tool that predicts fracture risk over 10 years, with a ≥3% risk for hip fractures and more than 20% for major osteoporotic fractures.[32,33]

Due to the limited availability of DEXA scanners in resource-constrained environments, FRAX® can be used to identify individuals who are at risk of osteoporotic fractures. When DEXA is not readily available, FRAX® is a cost-effective tool for predicting the risk of fractures.[34] Prednisolone raises the FRAX®- predicted risk of hip fracture by 20% and severe osteoporotic fracture by 15% if the dose is greater than 7.5 mg per day.[34]

In patients with type 2 diabetes mellitus (T2DM), FRAX® often underestimates fracture risk, who, despite higher BMD, face greater risk due to poor bone quality. Modifications to FRAX® are needed for accurate risk assessment.[35]

For a patient with T2DM, either of the following three changes should be considered:[35]

1. Rheumatoid arthritis can be used as a proxy for T2DM
2. Femoral neck BMD may be reduced by 0.5 units
3. The chronological age of the patient with T2DM to be increased by 10 years

2.4.5. Expert recommendation

Bone turnover markers (BTMs) are dynamic indicators, which can be used in monitoring treatment response or for failure to treatment or identify non-compliant patients, or secondary osteoporosis cases or if wrong t-score or z-score data, contingent upon availability and not done routinely.

2.4.5.1. Evidence

BTMs are valuable for assessing treatment response, guiding decisions and improving outcomes.[36] Additionally, they aid in determining the causes of secondary osteoporosis, especially in individuals who experience rapid bone loss and significant bone turnover.[37,38]

Serum CTX-1 tracks treatment response to antiresorptive therapy. For anabolic therapy, serum P1NP monitors response at baseline, after 1–3 months, 6 months and 12 months.

2.5. Treatment

2.5.1. Expert recommendation

Lifestyle modification should be initiated in patients who are at a low fracture risk, without prior fragility fractures.

2.5.1.1. Evidence

The UK clinical guideline for the prevention and treatment of osteoporosis strongly recommends lifestyle advice for men and women with a low fracture risk and no history of fragility fractures.[39] Lifestyle factors such as nutrition, alcohol, smoking, carbonated drinks, vitamin D deficiency and physical activity impact bone health. Weight-bearing exercises such as hiking, dancing and resistance training are especially beneficial for bone strength.[40]

2.5.2. Expert recommendation

Ensure sufficient calcium intake, with a total intake (from diet and supplements if necessary) of at least 1,000–1,200 mg/day in postmenopausal females with osteoporosis aged 50 years and older.

2.5.2.1. Evidence

To prevent osteoporosis and reduce the risk of fractures, the Institute of Medicine advises taking 1,000–1,200 mg of calcium each day.[41] According to the clinician's guide to osteoporosis prevention and treatment, males aged 50 to 70 should consume 1,000 mg of calcium daily, while women aged 51 and older should consume 1,200 mg.[42] Kindly note that daily intake of over 500 mg of elemental calcium is essential.[43]

2.5.3. Expert recommendation

Supplement with vitamin D3 if serum 25(OH)D levels are <30 ng/mL, i.e., insufficient or deficient of vitamin D. In such cases, a loading dose of 60,000 IU/week for 8 weeks, followed by 60,000 IU once a month, i.e., 2,000 IUs as a daily maintenance therapy, is typically required to maintain an optimal serum 25(OH)D level.

2.5.3.1. Evidence

A dose of 60,000 IU per week for 10 weeks boosts vitamin D levels by an average of 28.33 ng/mL.[44] The Indian Menopause Society guidelines (2019–2020) recommend maintenance therapy with either 2,000 IU/day of vitamin D supplements or a single annual intramuscular dose of 600,000 IU of cholecalciferol.[45] The use of collagen builders, high-protein diet and essential amino acids is also a necessary adjuvant before starting any anti-osteoporotic treatment.[46]

2.5.4. Expert recommendation

All individuals with osteoporosis should be initiated on vitamin D and maintain serum 25(OH)D levels between 30 and 60 ng/mL. Initiate activated vitamin D (active 1, 25[OH]D) or a similar analogue in patients with hypoparathyroidism and kidney disease.

2.5.4.1. Evidence

Experts from the Brazilian Society of Endocrinology and Metabolism define normal 25(OH)D levels as 20–60 ng/mL, with levels below 20 ng/mL indicating deficiency.[47] Maintaining 25(OH)D levels above 30 ng/mL benefits patients with osteoporosis. Activated vitamin D analogues are preferred option for those with hypoparathyroidism and chronic kidney disease (CKD).[48,49]

2.5.5. Expert recommendation

Anti-osteoporosis therapy should be initiated in those with high or very high risk of fragility fractures. As per the AACE guidelines, anti-resorptive therapy is preferred in those with a high risk of fragility fracture, while initiating anabolic therapy in patients with a very high risk of fractures.

2.5.5.1. Evidence

Pharmacological treatment is typically advised for patients who have experienced a fragility fracture as their risk of subsequent fractures is significantly elevated.[50] (**Table 3**) provides the classification of risk of fracture among patients with osteoporosis.

Table 3: Classification of fracture risk for osteoporosis[11,51]

Fracture risk category	**Criteria**
Very high fracture risk	Recent fracture (within the past 12 months)
	Fractures while on approved osteoporosis therapy
	Multiple fractures
	Fractures while on drugs causing skeletal harm (e.g., long-term glucocorticoids)
	Very low T-score (e.g., <−3.0)
	High risk for falls or history of injurious falls
	Very high fracture probability by FRAX® (e.g., major osteoporosis fracture > 30%, hip fracture > 4.5%)
	High fracture probability based on other validated fracture risk algorithms
High fracture risk	Diagnosed with osteoporosis but not meeting criteria for very high fracture risk as defined above
	T-scores between −1.0 and −2.5 and a history of fragility fracture of the hip or spine
	T-scores between −1.0 and −2.5 and a FRAX® 10-year probability of major osteoporotic fracture ≥20% or 10-year probability of hip fracture ≥ 3%
Low risk of fracture	No prior hip or spine fractures
	Bone mineral density (BMD) T-score at the hip and spine both above −1.0
	10-year hip fracture risk < 3%, and 10-year risk of major osteoporotic fractures < 20%

2.5.6. Expert recommendation

Consider anabolic therapy for patients who develop new fragility fractures despite being on anti-resorptive treatment.

2.5.6.1. Evidence

The Asia–Pacific consensus recommends anabolic therapy for osteoporosis and sarcopenia patients with new fractures or a persistent high fracture risk despite anti-resorptive treatment.[52]

2.5.7. Expert recommendation

The optimal treatment sequence for osteoporosis patients involves initiating anabolic therapy, followed by anti-resorptive therapy.

2.5.7.1. Evidence

Research indicates that greater results are obtained when osteoporosis is treated with an anabolic agent first, then an anti-resorptive medication. Switching to Teriparatide after an insufficient anti-resorptive response is not an effective use of anabolic therapy.[53,54]

2.5.8. Expert recommendation

Romosozumab is safe and has a dual mechanism of action (increased bone formation and reduced resorption) in postmenopausal women. As of December 2024, in Japan and Australia, Romosozumab is approved for men with osteoporosis.

2.5.8.1. Evidence

Romosozumab, a monoclonal antibody, inhibits sclerostin, activating the Wnt/β-catenin pathway. **Figure 2** illustrates the mode of action of Romosozumab.[55]

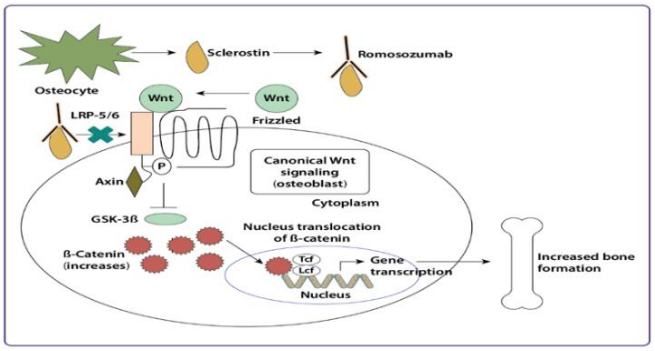

Figure 2: The mechanism of action of Romosozumab[96]

A systematic review of 25 randomised controlled trials (RCTs) (n = 24,942), treatment with Romosozumab, demonstrated no significant differences in cardiovascular mortality or major events as compared to placebo. This suggests that Romosozumab is a safe option for postmenopausal osteoporosis, with a lower cardiovascular risk. It is approved in Australia and Japan to improve BMD and lower fracture risk in men too.[56,57]

2.5.9. Expert recommendation

Romosozumab, as per the label, is recommended for a 12-month treatment course.

2.5.9.1. Evidence

In a real-world study, researchers observed that after 6 and 12 months of Romosozumab treatment, BMD increased significantly, by 7.4% and 12.2% at the lumbar spine, 1.8% and 5.8% at the total hip and 2.9% and 6.0% at the femoral neck ($p < 0.001$), respectively. In the treatment-naïve group, at 6 months and 12 months of Romosozumab treatment, lumbar spine BMD increased by 9.4% and 14.4%, respectively.[58] The recommended dose of Romosozumab is 210 mg administered subcutaneously once a month for 12 months.[59]

2.5.10. Expert recommendation

Romosozumab is contraindicated in patients with cardiac events or stroke in the previous one year, as per the current label.

2.5.10.1. Evidence

Romosozumab is safe for postmenopausal osteoporosis but is not recommended for those with a recent myocardial infarction or stroke due to cardiovascular risks observed in the ARCH and BRIDGE trials.[59] However, real-world data show fewer cardiovascular events compared to PTH analogues, with no significant differences in cardiovascular mortality or major events versus placebo, indicating a better risk profile.[60,61]

2.6. Indication

2.6.1. Expert recommendation

The ideal patient profile in postmenopausal women for initiating Romosozumab includes the following:

1. Patients requiring a lowering in vertebral, hip and non-vertebral fracture risk who are experiencing a decline in BMD despite anti-resorptive therapy
2. Patients who have not responded to or are not improving on PTH therapy
3. Patients at a very high risk of fractures with a significant risk of falls
4. Patients not responding to PTH
5. Patients with CKD at a very high risk of fragility fracture

2.6.1.1. Evidence

The ARCH trial showed Romosozumab's superior efficacy over Alendronate in postmenopausal women with severe osteoporosis. After 1 year, Romosozumab increased BMD at the spine by 2.5 times and at the hip by two times compared to Alendronate, reducing vertebral fracture risk by 37% in year 1 and 48% in year 2.[62]

A phase 3 study showed that after 1-year, Romosozumab (210 mg monthly) raised total hip BMD by 2.6%, whereas Teriparatide (20 μg daily) decreased it by 0.6%, showing a 3.2% difference ($p < 0.0001$), favouring, Romosozumab in postmenopausal females with osteoporosis.[63]

A meta-analysis by Möckel L *et al*. demonstrated that sequential treatment with Romosozumab followed by an anti-resorptive medication lowered the risk of falls by 12% vs. controls.[64]

Tian A *et al*. in a systematic observed that Romosozumab significantly lowered BMD at the lumbar spine, hip and femoral neck vs. Teriparatide, with fewer injection-site reactions.[65]

The evidence suggests that Romosozumab is effective for postmenopausal females with osteoporosis and mild-to-moderate kidney impairment, with a consistent safety profile across various kidney function levels.[66]

2.6.2. Expert recommendation

Romosozumab can be a promising therapy in postmenopausal women with glucocorticoid-induced osteoporosis who are at a very high risk of fracture.

2.6.2.1. Evidence

A recent study comparing Romosozumab and Denosumab in high fracture risk glucocorticoid users found that Romosozumab led to significantly greater spine BMD gains at 12 months (+7.3% vs. +2.3%; $p < 0.001$) and 24 months (+9.7% vs. +3.0%; $p < 0.001$). Hip BMD gains were similar, although injection-site reactions were more frequent with Romosozumab.[67]

2.6.3. Expert recommendation

Romosozumab is not to be combined with or followed by Teriparatide.

2.6.3.1. Evidence

Romosozumab promotes bone formation and prevents resorption, making its combination with other osteoporosis treatments unnecessary and unstudied.[59] To date, no clinical evidence is available on combining Romosozumab with Teriparatide or using them sequentially.

2.7. Denosumab for management of osteoporosis

2.7.1. Expert recommendation

Denosumab can be initiated in patients with a high risk of fragility fracture due to glucocorticoid-induced osteoporosis.

2.7.1.1. Evidence

In May 2018, Denosumab was approved for glucocorticoid-induced osteoporosis in high fracture risk men and women. A systematic review and meta-analysis by Yanbeiy ZA showed that Denosumab outperforms bisphosphonates in improving lumbar spine and hip BMD in patients with glucocorticoid-induced osteoporosis, making it a viable treatment option.[68]

2.7.2. Expert recommendation

Long-term treatment with Denosumab for up to 10 years has been shown to be associated with continued BMD gain for postmenopausal women with osteoporosis without a plateau effect.

2.7.2.1. Evidence

In the 10-year FREEDOM Extension study, Denosumab showed continuous BMD increase, sustained low vertebral fractures and reduced non-vertebral fracture risk. Rates of osteonecrosis of the jaw and atypical femoral fractures remained low, demonstrating Denosumab's favourable benefit/risk profile. Unlike bisphosphonates, Denosumab continues to increase bone density beyond 3–4 years.[69]

2.7.3. Expert recommendation

The use of Denosumab may be advocated for the treatment of osteoporosis in patients with CKD across different estimated glomerular filtration rates (eGFR), considering its relative safety profile, except when the patient needs to be monitored for hypocalcaemia in advanced CKD and dialysis.

2.7.3.1. Evidence

A study by Jamal SA *et al*. found that Denosumab reduced fracture risk and improved BMD compared to placebo, with no significant efficacy differences across eGFR subgroups. Changes in creatinine, calcium and adverse events were comparable between groups and did not depend on kidney function.[70]

2.7.4. Expert recommendation

The combination of Denosumab and Teriparatide, considered for postmenopausal female with very high risk or imminent fracture, has demonstrated to increase BMD more compared to either agent alone.

2.7.4.1. Evidence

A study by Tsai JN showed that after 15 months, the 40 μg combination of Denosumab and high-dose Teriparatide significantly increased spine areal BMD (aBMD) (17.5% ± 6.0) compared to the 20-μg group (9.5% ± 3.2, $p < 0.0001$). The 40-μg group also had greater improvements in femoral neck (6.8% vs. 4.3%) ($p = 0.04$) and total hip aBMD (6.1% vs. 3.9%), with both differences statistically significant ($p < 0.0001$).[71]

At 24 months, the combination group showed the greatest BMD increases: 12.9% ± 5.0% at the lumbar spine, 6.8% ± 3.6% at the femoral neck and 6.3% ± 2.6% at the total hip, all significantly higher than the individual drug groups ($p < 0.001$).[72]

2.7.5. Expert recommendation

Switching from Teriparatide to Denosumab increases BMD, whereas switching from Denosumab to Teriparatide can lead to bone loss.

2.7.5.1. Evidence

In an RCT, postmenopausal females switching from Teriparatide to Denosumab showed continued BMD increase, whereas switching from Denosumab to Teriparatide led to bone loss, particularly at the hip. These findings should guide treatment decisions for postmenopausal osteoporosis, especially regarding fracture risk.[73]

2.7.6. Expert recommendation

Anti-osteoporosis pharmacotherapy should be considered in patients post-operatively after surgery to enhance BMD and continued lifelong.

2.7.6.1. Evidence

Evidence suggests a positive impact of anti-osteoporosis pharmacotherapy in patients after surgery. Osteoanabolic agents are recommended for patients at very high fracture risk, such as those with previous osteoporotic fractures or poor bone health, particularly when undergoing bone-related surgery.[74]

In a review article, the researcher evaluated the impact of anti-resorptive agents on the outcomes and durability of total hip and knee arthroplasty. The study included 24 studies on total hip replacement, eight on total knee replacement and two covering both procedures. Most of the studies focused on oral bisphosphonates, showing significant improvements in BMD after 6 months of postoperative anti-resorptive therapy, with better outcomes and lower revision rates in cemented total knee arthroplasty.[75]

Evidence indicates that anabolic treatment can be used for patients undergoing fragility fracture and non-union surgery, and anti-resorptive treatment can be used for patients undergoing replacement or fixation surgery.[76]

3. Conclusion

Osteoporosis is a silent, chronic condition that warrants early diagnosis and lifelong management, such as diabetes and hypertension. Screening is recommended for postmenopausal female more than 50 years of age, male more than 60 years of age and high-risk individuals with chronic illnesses, long-term medication use, fragility fractures, smoking or testosterone deficiency. Diagnosis primarily relies on DEXA scans, with lateral spine x-rays as alternatives when DEXA is unavailable.

The guidelines suggest that osteoporosis management involves adequate calcium intake and maintaining vitamin D levels through supplementation**.** Anti-osteoporosis therapy should be initiated in patients with high or very high risk of fragility fractures. Anti-resorptive therapy is suitable for high-risk patients, whereas anabolic therapy, including Romosozumab, is recommended for very high-risk cases. The ideal patient profile in postmenopausal women for initiating Romosozumab includes patients requiring reduction in vertebral, hip and non-vertebral fracture risk who are experiencing a decline in BMD despite anti-resorptive therapy, patients who have not responded to or are not improving on PTH therapy, patients at very high risk of fractures with a significant risk of falls, patients not responding to PTH and patients with CKD at very high risk of fragility fracture.

Romosozumab may be a treatment option for postmenopausal women with glucocorticoid-induced osteoporosis who are at very high risk of fracture. In addition, Denosumab can be initiated in patients at high risk of fragility fractures due to glucocorticoid-induced osteoporosis.

Post-operative pharmacotherapy should enhance bone healing and fracture prevention.

A personalised, risk-based approach integrating pharmacologic therapy, calcium-vitamin D optimisation and lifestyle modifications remains the cornerstone of osteoporosis management.

These expert-reviewed and recommended statements serve as a guide for healthcare providers and aim to optimise patient care and outcomes.

4. Ethical Approval

Not needed.

5. Author Contributions

All authors have made significant contributions to the conception of the work, the acquisition, analysis and interpretation of data, as well as drafting and substantially revising the work.

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7. Conflict of Interest

The authors declare no conflicts of interest.

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