Management and Prevention of Osteoporosis and Fragility Fractures: Key Practice Points

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Introduction

Osteoporosis is a significant non-communicable disease and the most prevalent bone disorder, affecting one in three women and one in five men over the age of 50 globally.[1-2] In India, the prevalence of osteoporosis is particularly high, with an overall prevalence of 24.7% among men and women aged 30–90 years, and a higher prevalence among women from low-income groups, where 52% experience osteopenia and 29% suffer from osteoporosis.[3] Osteoporosis affects an estimated 200 million women globally, particularly as they age, and is notably underdiagnosed and undertreated in Asia, especially in rural areas where fractures are often managed conservatively. The aging population is expected to increase the incidence of osteoporosis in postmenopausal women significantly.[1]

A systematic review found that postmenopausal women in India are at significant risk of low bone mineral density (BMD), with a 29% prevalence of osteoporosis in the lumbar spine region, 6% in the hip region, and 29% in the femoral neck region. Osteopenia was prevalent in 37% of women in the lumbar spine and femoral neck, and 6% in the hip.[4]

Thus, addressing postmenopausal osteoporosis is crucial because women in this stage of life spend approximately one-third of their lives with a reduced bone mass and an elevated risk of fractures. Fractures of the pelvis, vertebrae, and distal radius contribute significantly to morbidity and mortality, with a 20% mortality rate within the first year following a hip fracture.[5]

Objectives

The objective of this expert opinion is to offer practice points based on the latest evidence for diagnosing and managing osteoporosis, as well as preventing fragility fractures in postmenopausal women, and in men aged 50 years or older. Additionally, it aims to address gaps in medical care related to managing fragility fractures in the Indian context.

Method

A core committee was formed, comprising a diverse, multidisciplinary group of experts, including rheumatologists, endocrinologists, orthopedic surgeons, gynecologists, general practitioners (GPs), physicians specializing in elderly care, and patients with osteoporosis. A panel meeting was held where each topic was presented and discussed, and a consensus was reached. Thus, this document provides much-required insights and useful, practical, and accurate feasible guidance that aids a practicing clinician across the country.

Definitions

Osteoporosis

Osteoporosis is a systemic skeletal disorder marked by reduced bone density and a decline in bone quality (microarchitectural changes),

resulting in weakened bone strength and an elevated risk of fractures from minimal trauma.[6-7]

Operational Definition of Osteoporosis by WHO

World Health Organization cutoffs used in the diagnosis of osteoporosis (BMD at the hip)	
Normal Bone	T-score > −1 SD
Osteopenia	T-score between −1 and −2.5 SD
Osteoporosis	T-score < −2.5 SD
Established (severe) Osteoporosis	T-score ≤ −2.5 SD with a history of fragility fractures

The World Health Organization (WHO) defines osteoporosis as a BMD that is 2.5 standard deviations (SD) or more below the average BMD for young, healthy women, as measured by dual-energy X-ray absorptiometry (DEXA). This definition applies to post-menopausal women and men aged 50 years or older, with a T-score of ≤ −2.5 SD (Table 1).[6]

Fragility fractures

The application of mechanical stress exceeding the bone's structural capacity results in osteoporotic fractures. The vertebral body, proximal femur, proximal humerus, and distal radius are the most affected sites. The WHO defines fragility fractures as those resulting from low-energy trauma, such as a fall from a standing height, which would not typically result in a fracture under normal circumstances. Skeletal fragility is characterized by both reduced bone density and impaired bone quality. This includes alterations in bone architecture, geometry, and the microstructural properties of bone components such as collagen and minerals, as well as the presence of microdamage. The risk of low-trauma fractures increases with age in both sexes.[6]

When evaluating a patient, certain clinical history details may indicate a vertebral fracture. These include recent trauma, prolonged corticosteroid use, advanced age, structural spinal deformities, height loss greater than 6 cm, and a reduced distance between the last rib and the iliac crest (less than 2 fingers). Therefore, it is important to thoroughly assess for symptoms such as dorso-lumbar pain, progressive height loss, or dorsal kyphosis, as these may lead to changes in respiratory or gastrointestinal function.[7]

Risk factors

Various risk factors that contribute to postmenopausal osteoporosis are broadly classified as non-modifiable or modifiable risk factors. Non-modifiable risk factors for osteoporosis include sex, age, ethnicity, and genetics. Women, especially in India, are at higher risk due to smaller body frames, lower calcium intake, and inadequate sunlight exposure. Estrogen deficiency from early menopause (average age 46.2 years in India) significantly contributes to osteoporosis. Studies show that osteoporosis prevalence increases with age, particularly among Indian women. Additionally, genetic factors, race, and ethnicity affect peak bone mass, with Asian Indian women having 5%-15% lower BMD compared to non-Asian women. Variations in vitamin D receptor genes also contribute to ethnic differences in BMD.[8]

Modifiable risk factors for osteoporosis include:

- **Nutritional factors:**
  - » **Calcium and Vitamin D**: Indian diets often lack sufficient calcium and vitamin D, especially in lower socioeconomic classes. Poor dairy intake, high phytate content, indoor lifestyles, and traditional clothing contribute to deficiencies, which hinder bone health.
- **Nutritional Status**: Low body weight (<60 kg) and poor nutrition increase the risk of osteoporosis. Low BMI and sarcopenia are associated with a reduced BMD.
- **Lifestyle Factors**: Sedentary lifestyles, reduced sun exposure, and lack of physical activity, particularly weight-bearing exercise, contribute to lower BMD. Smoking, alcohol use, and diabetes further increase fracture risk.
- **Medication Use**: Long-term use of glucocorticoids, proton pump inhibitors, anticonvulsants, and minimal hormone replacement therapy are significant contributors to osteoporosis.[8]

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Bone cement augmentation

- The WHO defines of osteoporosis based on a T-score of ≤ −2.5 SD, measured by DEXA at the hip, with a T-score ≤ −2.5 SD indicating osteoporosis, and ≤ −2.5 SD with fragility fractures indicating severe osteoporosis.
- Fragility fractures should be considered a hallmark of osteoporosis, typically occurring in the vertebral body, proximal femur, proximal humerus, and distal radius due to low-energy trauma.
- Non-modifiable risk factors for osteoporosis should be recognized, including sex, age, ethnicity, and genetic predisposition. Indian women are particularly at risk due to smaller body frames, lower dietary calcium intake, and earlier menopause.
- Modifiable risk factors for osteoporosis should be addressed, such as inadequate calcium and vitamin D intake, low body weight, physical inactivity, smoking, excessive alcohol consumption, and the use of medications like glucocorticoids and proton pump inhibitors.

Diagnosis of Osteoporosis

Clinical Assessment:

Osteoporosis should be considered in any adult with a fragility fracture. For postmenopausal women, a height loss greater than 4 cm could suggest vertebral fractures. Persistent back pain may also indicate underlying vertebral fractures.[8]

Dual-Energy X-Ray Absorptiometry (DXA):

DXA is the main method for measuring BMD. BMD is reported as T-scores, comparing an individual's BMD to a young adult reference population, and Z-scores, comparing it to age-matched peers. In India, Caucasian female data is used for T-scores due to limited local data. DXA helps assess fracture risk and track treatment response.[8]

Indications for DXA: Perform DXA for women aged 60 and older, and men aged 65 and older, regardless of risk factors. Consider DXA for postmenopausal women under 60 and men aged 50-64, if they have risk factors. It is also needed for those with fragility fractures before age 50, or those with conditions or medications affecting bone mass. Early screening may be necessary due to the higher fracture prevalence in Indians.[8]

Biochemical Investigations:

Before starting treatment, check serum calcium, phosphorus, alkaline phosphatase, creatinine, 25-hydroxyvitamin D, and intact parathyroid hormone (iPTH). For secondary osteoporosis, perform additional tests based on clinical findings.[8]

Bone Turnover Markers (BTMs):

BTMs show short-term changes in bone remodeling but are not used for diagnosis. They are useful for monitoring the effectiveness of osteoporosis treatments. Measure BTMs before starting therapy for comparison during follow-up.[8]

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diagnosis of osteoporosis

- Osteoporosis should be considered in adults with fragility fractures, and height loss greater than 4 cm or persistent back pain in postmenopausal women should suggest possible vertebral fractures.
- DXA should be the primary method for assessing BMD, with results reported as T-scores and Z-scores to evaluate fracture risk and monitor treatment response.
- DXA should be performed for women aged 60 and older and men aged 65 and older. It should be considered earlier for postmenopausal women under 60 and men aged 50–64 with risk factors, as well as for individuals with fragility fractures or conditions affecting bone mass.
- Serum calcium, phosphorus, alkaline phosphatase, creatinine, 25-hydroxyvitamin D, and iPTH should be evaluated before starting treatment, with additional tests for secondary osteoporosis as indicated.
- BTMs should not be used for diagnosis but should be measured before starting therapy to monitor treatment effectiveness during follow-up.

Indications for treatment

The initiation of anti-osteoporotic treatment should rely on clinical screening tools like the Fracture Risk Assessment Tool (FRAX) and imaging methods such as DXA scans or plain radiography. FRAX evaluates the 10-year risk of major osteoporotic fractures (including wrist, vertebral, hip, and shoulder) based on various risk factors, with treatment thresholds differing across ethnic groups. The Endocrine Society guidelines recommend treatment for postmenopausal women at high fracture risk, particularly those with recent fragility fractures. The primary objective of starting osteoporosis treatment is to minimize the incidence of fragility fractures, which can lead to significant morbidity, mortality, and economic costs. Key indications for therapy initiation are outlined in a summarized format in Table 2.[8]

Table 2. Summarizing the key indications for initiating anti-osteoporotic therapy
• A vertebral fracture (clinically apparent or found on vertebral imaging) or non-vertebral fracture (hip, wrist, and humerus).
• In individuals >50 years of age with T-score ≤ −2.5 at the femoral neck or total hip or lumbar spine measured by DXA.
• In individuals with osteopenia (T-score between−1.0 and−2.5 at the femoral neck or lumbar spine) with clinical risk factors or a 10-year probability of a hip fracture ≥3.5% or a 10-year probability of a major osteoporosis-related fracture ≥10.5% based on the FRAX tool (based on limited data in Indians).
• In individuals with type 2 diabetes mellitus, the intervention threshold should be increased to T-score ≤ −2.0 at the femoral neck or total hip or lumbar spine measured by DXA.

Fundamentals of osteoporosis management

Nonpharmacological measures for osteoporosis prevention and treatment[8]

- Maintain serum 25-hydroxyvitamin D (25[OH] D) levels at or above 20 ng/mL, ideally between 30–40 ng/mL.
- Supplement with vitamin D3 if needed; typically, 1000 to 2000 international units (IU) daily are required for optimal levels in Indians. Adjust doses for factors like obesity, malabsorption, or older age.

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initiating anti-osteoporotic treatment

- Anti-osteoporotic treatment should be initiated based on clinical screening tools like the FRAX score and imaging methods such as DXA scans or plain radiography.
- FRAX should be used to evaluate the 10-year risk of major osteoporotic fractures, with treatment thresholds varying by ethnicity.
- Treatment should be initiated in postmenopausal women at high fracture risk, especially those with recent fragility fractures, as recommended by the Endocrine Society guidelines.
- Therapy should be considered for individuals over 50 with a T-score ≤ −2.5 at key skeletal sites (femoral neck, total hip, or lumbar spine), those with osteopenia and additional risk factors, or a significant FRAX score, and in those with type 2 diabetes with a T-score ≤ −2.0.

- Ensure a total calcium intake of at least 1000 mg/day from both diet and supplements for women aged 50 and older.
- Limit alcohol consumption to no more than 2 units per day.
- Encourage smoking cessation.
- Promote an active lifestyle that includes weight-bearing and balance exercises.
- Guide on reducing fall risk, particularly for older patients.

Pharmacologic Therapies

Pharmacologic treatments for osteoporosis fall into two main categories: antiresorptive agents, which inhibit bone resorption by osteoclasts, and anabolic agents, which stimulate bone formation by osteoblasts. Both types of drugs have been proven to enhance BMD and lower fracture risk. Table 3 summarizes the medications approved by the Food and Drug Administration (FDA) for the treatment of osteoporosis.[9]

Table 3. Drugs approved by the Food and Drug Administration for the treatment and prevention of osteoporosis				
Drug class and agent	Method and frequency of administration	Type of fracture risk reduction	Side effects	Approved use for osteoporosis
Bisphosphonates				
Alendronate	Oral: 35–70 mg/week	Vertebral, nonvertebral, hip	Common: esophagitis, musculoskeletal symptoms; Rare: Osteonecrosis of the Jaw (ONJ), atypical femur fractures	Treatment and prevention
Risedronate	Oral: 35 mg/week or 150 mg/month (single dose or two 75 mg doses on consecutive days)	Vertebral, nonvertebral, hip	Common: esophagitis, musculoskeletal symptoms; Rare: ONJ, atypical femur fractures	Treatment and prevention
Ibandronate	Oral: 150 mg/week; Intravenous: 3 mg every 3 months	Vertebral	Common: First-dose (intravenous) reaction, esophagitis, musculoskeletal symptoms; Rare: ONJ, atypical femur fractures	Treatment and prevention
Zoledronic acid	Intravenous: 5 mg/year	Vertebral, nonvertebral, hip	Common: Acute-phase response (most often after first dose), musculoskeletal symptoms; Rare: ONJ, atypical femur fractures	Treatment and prevention
Anabolic: Teriparatide	Subcutaneous: 20 µg/day	Vertebral, nonvertebral	Common: Nausea, leg cramps; Rare: Hypercalcemia, osteosarcoma	Treatment
Biologic: Denosumab	Subcutaneous: 60 mg every 6 months	Vertebral, nonvertebral, hip	Common: Cellulitis or skin reactions; Rare: ONJ, atypical femur fractures	Treatment
Calcitonin	Intranasal: 200 IU/day	Vertebral	Nasal congestion	Treatment
SERM: Raloxifene	Oral: 60 mg/day	Vertebral	Venous thromboembolism, hot flashes, leg cramps, nausea	Treatment and prevention
Estrogens			Venous thromboembolism, increased risk of breast cancer and cardiovascular disease	Prevention
Conjugated equine estrogen	Oral: 0.15–1.25 mg/day	Vertebral, nonvertebral, hip		
17β-estradiol	Oral: 0.025–0.10 mg/day; Transdermal: 2 times/week	No data from randomized trials		
Ultra-low-dose 17β-estradiol	Oral: 0.014 mg/day	No data		

Menopausal Hormone Therapy[10-11]

- Estrogen-progesterone therapy (EPT) or estrogen therapy (ET) may be used for osteoporosis treatment in early postmenopausal women with vasomotor symptoms, unless contraindicated. In patients with an intact uterus, progestin should be added, either daily or cyclically, to protect against endometrial stimulation.
- Menopausal hormonal therapy (MHT) requires individualized dosing, annual risk–benefit assessments, and regular gynecological exams. Monthly self-breast exams and annual clinical exams are recommended, with mammograms every 1–3 years.
- All estrogen preparations, including low-dose and non-oral routes, effectively preserve bone mass. In cases of hypertriglyceridemia, obesity, glucose intolerance, or tobacco use, non-oral routes should be preferred.
- MHT should not be initiated solely for bone protection after 10 years post-menopause. Extended MHT may be considered for women with reduced bone mass, based on a risk–benefit assessment.
- MHT is the primary therapy to prevent bone loss in premature menopause and secondary amenorrhea.
- Progestogens should be added to estrogen therapy in women with a uterus.
- MHT given to women below 60 or within 10 years of menopause carries minimal risks. Risk-benefit tables can assist in counseling.
- Tibolone, preferred for symptomatic women with dense breast tissue, and SERMs like raloxifene and bazedoxifene (BZA), effectively reduce vertebral fracture risk, maintain bone health, and lower breast cancer risk. While

raloxifene and estrogen increase VTE risk, BZA benefits bone and lipid profiles without affecting hot flashes. Conjugated estrogens/bazedoxifene (CE/BZA) is FDA-approved for vasomotor symptoms and osteoporosis prevention, though not yet available in India.

Bisphosphonates

- In postmenopausal women at high risk of fractures, initial treatment with bisphosphonates (alendronate, risedronate, zoledronic acid, and ibandronate) is recommended to reduce fracture risk.[12]
- Bisphosphonates are recommended as the first-line drugs for treating postmenopausal women, with proven efficacy in the prevention of vertebral and nonvertebral fractures, including hip fractures.[10]
- Alendronate, denosumab, risedronate, and zoledronate are approved agents with efficacy to reduce hip, nonvertebral, and spine fractures as initial therapy for most osteoporotic patients with high fracture risk.[11]
- For oral bisphosphonates, a bisphosphonate holiday after 5 years of treatment should be considered if fracture risk is no longer high (such as when the T score is greater than -2.5, or the patient has remained fracture-free), but continue treatment up to an additional 5 years if fracture risk remains high.[8]

Teriparatide

Teriparatide (TPTD), a recombinant human parathyroid hormone, has been a leading anabolic treatment for severe osteoporosis since its pivotal clinical trial 20 years ago. Administered as a daily subcutaneous injection, TPTD significantly increases BMD, particularly in the spine, and is highly effective in reducing vertebral fractures. The treatment stimulates bone formation by increasing osteoblast activity, leading to enhanced bone mass and strength. It is especially beneficial for patients with severe spinal osteoporosis, where it has demonstrated superior efficacy in preventing vertebral fractures compared to bisphosphonates like Risedronate. After completing TPTD therapy, continuing with antiresorptive treatment is recommended to sustain the bone gains.[13]

- Teriparatide is recommended for post-menopausal women and men with severe osteoporosis (T-score ≤ -2.5) and prevalent fractures.[8]
- The Endocrine Society Guideline 2020 recommends teriparatide or abaloparatide treatment for up to 2 years for the reduction of vertebral and nonvertebral fractures in postmenopausal women with osteoporosis at very high risk of fracture, such as those with severe or multiple vertebral fractures.[12]
- The American Association of Clinical Endocrinologists' Guidelines suggest limiting treatment with abaloparatide and teriparatide to 2 years and continuing with a bisphosphonate or denosumab to maintain bone density gains.[11]

Denosumab

- Denosumab, a recently approved monoclonal antibody in India, specifically targets RANKL and is used for postmenopausal women with osteoporosis who are at high risk of fractures.[10]
- Denosumab increases trabecular and cortical bone strength, reduces vertebral, nonvertebral, and hip fracture risk, and improves BMD more than bisphosphonates. It provides long-term benefits over 10 years without requiring drug holidays.[10]
- Administered as a 60 mg SC injection every 6 months, denosumab is well-tolerated, even in patients with creatinine clearance <30 mL/min, where bisphosphonates and teriparatide are contraindicated.[10]
- In postmenopausal women with osteoporosis on denosumab, fracture risk should be reassessed every 5 to 10 years. If high fracture risk persists, denosumab should be continued or replaced with other osteoporosis therapies.[12]

- Denosumab should not be delayed or stopped without initiating subsequent antiresorptive therapy (e.g., bisphosphonates, hormone therapy, or selective estrogen receptor modulators) to prevent a rebound in bone turnover and reduce the risk of rapid bone loss and fractures.[12]

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the use of pharmacotherapy for osteoporosis and prevention of fragility fractures

- Estrogen-progesterone or estrogen therapy should be used for osteoporosis in early postmenopausal women with vasomotor symptoms, with progestin added if the patient has an intact uterus.
- Bisphosphonates should be used as the initial therapy for postmenopausal women at high fracture risk, effective in reducing vertebral, nonvertebral, and hip fractures.
- A bisphosphonate holiday should be considered after 5 years if fracture risk is low, but treatment should continue up to an additional 5 years, if fracture risk remains high.
- Teriparatide should be considered for patients with severe spinal osteoporosis due to its significant efficacy in increasing spinal BMD and reducing vertebral fractures.
- Teriparatide should be used for up to 2 years in severe osteoporosis with prevalent fractures, followed by bisphosphonates or denosumab to maintain bone density gains.
- Denosumab should be used for postmenopausal women at high fracture risk, increasing bone strength and reducing fracture risk over 10 years without drug holidays.
- Denosumab should not be stopped or delayed without initiating subsequent antiresorptive therapy to prevent a rebound in bone turnover and increase in fracture risk.

Principles of fracture fixation

The principles of fracture fixation are:[14]

1. Fracture reduction to restore anatomical relationships.
2. Fracture fixation to provide absolute or relative stability as the "personality" of fracture, patient, and injury requires.
3. Preservation of blood supply to soft tissues and bone.
4. Early and safe mobilization of the injured part and the patient.

Challenges encountered in the treatment of osteoporotic fractures

Immobilization with the use of conservative measures (cast or orthosis) or surgical fixation with osteosynthesis implants (screws, plates, or nails) is necessary to promote healing of the fractured bone. Further, in this context, osteoporotic bone treatment poses a challenge for several reasons.[15]

Nature of osteoporotic bone: Decreased density and increased brittleness of osteoporotic bones are prone to fracture into many smaller individual fragments, leading to more complex fractures than healthy bone. Therefore, considerable surgical skills for reduction of the fractures and efficient implants for stabilization are essential.

Osteoporotic fractures in the elderly: Age-associated reductions in sense of balance, coordination, and proprioception with reduced vision in the elderly results in uncoordinated limb loading and increased risk of falling. Therefore, a fracture fixation that can provide maximum stability to withstand full weight bearing may be essential.

Further, the elderly have a high prevalence of comorbidities which makes them more vulnerable to post-traumatic complications and necessitates quick and minimally invasive surgery as well as rapid mobilization. Hence, sufficient mechanical stability to immediately mobilize the patient should be the main aim of fracture fixation in this population.[15]

The osteoporotic bone also has deteriorated mechanical properties that reduce resistance to loading by rigid osteosynthesis implants, which may cause implant loosening, implant cutout, and peri-implant fractures.[15]

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principles of fracture fixation

While treating osteoporotic fractures, the nature of osteoporotic bone, age of the patient and associated sense of balance, coordination, and reduced vision, comorbidities, and risks of complications should be considered. This can help the clinician in deciding the appropriate surgical technique and impant to provide efficient stabilization, and immediate mobilization of the patient post-fracture fixation.

Load sharing devices

Internal fixation devices that support the load bearing with bone should be considered to reduce stress at the bone-implant interface. The load-sharing devices that are suitable for osteoporotic bone include sliding nail plate devices, intramedullary nails, and tension band constructs.[16]

Plate fixation: Locking vs. conventional compression plates

Bone-related changes due to aging and osteoporosis reduce the strength of internal fixation thereby affecting the holding power of the plate-screw construct to the bone. Reports have shown that the screw's holding power decreases by 50% with a reduction in cortical thickness of 1 mm.[17]

With the use of conventional plates, the load is transferred from the bone to the plate across the fracture area and back to the bone again. Compressing the plate onto the bone by tightening the screws produces friction in conventional plating, which induces preload on the bone around the screws and increases the risk of screw pullout.[17]

Whereas with locking plates, the load is transferred through the screws and the interface between the screw and plate, which prevents individual screws from toggling and cutting through the bone.[18]

A study compared the mechanical behavior of the locking compression plate (LCP) device, with the traditional dynamic compression plates (DCPs) in the bone of varying quality. The strain produced by both screw types within the bone was similar in healthy bone with a reduced fracture gap, but the DCP produced much larger strains in osteoporotic bone. It showed a significantly lower strain at the screw-bone interface when locking plates were used in osteoporotic bone. This shows that the use of locking plates was associated with improved performance in poorer bone quality.[18]

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locking plates

Locking plates can be considered as a suitable choice over conventional compression plates, as they allow screws to be threaded into the plate, creating a fixed-angle device, and reduces friction between the bone and plate. Therefore, locking plates can provide more stability with improved performance in cases of osteoporotic fractures.

Intramedullary nailing

The gold standard of treatment for diaphyseal long bone fractures is the intramedullary nails. They are specifically used in metadiaphyseal fractures of the distal femur, proximal tibia, and proximal humerus. These nails are located centrally within the bone, which shares the load and allows for healing through relative stability, secondary bone healing, and callus.[19] This central location placement and healing pattern are advantageous in the osteoporotic bone as it preserves the blood supply, fracture hematoma, and soft tissues in the fracture zone which facilitates fracture healing.[19-20]

The use of blades instead of screws or angle-stable locking screws is advantageous since they have a greater load-bearing surface and therefore higher stability.[20]

A biomechanical study showed that interlocking with a bladelike device was 41% stiffer ($p = 0.01$) and 20% stronger ($p = 0.02$) than conventional locking bolts under axial load.[21]

Another study has shown that patients with the angular stable locking option of intramedullary

nails showed significantly higher stiffness values and reduced fracture gap motion than those with conventional locked nails.[22]

Supplemental long plate fixation along with intramedullary fixation

Although intramedullary fixation is considered the standard form of long-bone surgical fixation, it is associated with complications such as implant breakage, implant cut-out, long bone fracture, nonunion, and rod bending. Hence, supplemental fixation techniques should be considered to reduce the risk of surgical implant failure of intramedullary devices. In addition to an intramedullary device in the long bone segment, a plate and screw construct at a fracture site is one such form of supplemental fixation.[23]

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intramedullary nailing

- Intramedullary devices can be considered suitable for the treatment of diaphyseal and metaphyseal fractures of the osteoporotic bone. Interlocking with a bladelike device should be preferred over conventional locking bolts as they are associated with a greater load-bearing surface and higher stability.
- Supplemental fixation techniques should be considered to reduce the risk of surgical implant failure of intramedullary devices.

Bone cement augmentation

Bone cement augmentation aims to improve implant anchorage and allow early mobilization. Acrylic bone cement such as polymethylmethacrylate (PMMA) has played an important role in orthopedic surgery.[24] It has been used for the fixation of prosthetic implants and neoplastic and vertebral fracture repair. Further, improvement in implant anchorage has been demonstrated by using calcium phosphate cement to augment the central void in the humeral head in fracture fixation of the proximal humerus.[17]

Osteoporotic fracture fixation augmentation may also involve allograft fibulas, which can be used as reduction tools as well as medial calcar support in comminuted proximal humerus fractures.[19]

The commonly used bone cement such as PMMA is nondegradable, which may cause complications, such as intramedullary hypertension and pulmonary embolism. Therefore, newer materials with good biodegradability can play an indispensable role in tissue repair and regeneration. Such biodegradable materials provide support, osteoconductivity, and osteoinduction at the implantation site, and may assist in bone tissue repair.[25]

Studies have shown that bone cement augmentation in the treatment of fractures significantly increased the number of cycles until failure.[26-27]

A systematic review was conducted to evaluate the effects of cement augmentation of intramedullary devices as an adjunct to internal fixation of trochanteric hip fractures. Cement augmentation was reported to be a safe and effective method of fixation to treat trochanteric fractures.[28]

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Bone cement augmentation

Bone cement augmentation is found to be a safe and effective approach for fixation, and should be considered in patients with osteoporotic fractures to improve implant anchorage and allow early mobilization.

Post-operative care of patients with osteoporotic fractures[29]

As the risk of a second fracture is highest in the early post-fracture period, immediate anti-osteoporosis treatment is recommended, using medications on time, in dosage, and on schedule as prescribed by the physician, and not stopping the medication without authorization.

It is recommended that drugs that cause accelerated bone loss, such as glucocorticoids be avoided as much as possible.

Patients with osteoporotic fractures should be monitored to prevent refracture; annual DXA bone density testing, bone turnover index testing every 3–6 months as appropriate, and periodic review to determine maintenance or adjustment of the treatment regimen.

Early activity is required after osteoporotic fracture surgery, preferably starting on the first postoperative day. Exercise should be gradual, and where their condition permits. Including a diverse range of physical activities can be beneficial, such as aerobic exercise, activities that build skeletal muscle strength, and balance training.

All postmenopausal women and men aged ≥50 years should undergo a multifactorial fall risk assessment to prevent the risk of falls.

Calcium and vitamin D intake:

- For patients with osteoporotic fractures, calcium intake should be increased appropriately, and it is recommended that at least 1,000 mg of calcium should be consumed every day.
- For over 50 years of age and postmenopausal women, 1,000 mg of calcium every day should be consumed, preferably from dietary intake, with the addition of calcium supplements, as appropriate.
- For people over 70 years of age, the recommended dietary intake of calcium is 1,200 mg per day (3 servings of dairy products or equivalent).
- Elderly people can be supplemented with active vitamin D for the prevention and treatment of osteoporosis with a dosage of is 800 to 1200 IU per day.
- The recommended dietary intake of vitamin D for elderly people over 70 years of age is 800 IU/d, and the maximum intake that can be tolerated is 4000 IU.

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