Contributors:
Prof. Dr. Suchitra N. Pandit
Consultant - Kokilaben Dhirubhai Ambani Hospital and research centre
Senior Vice President – Mumbai Society of Obstetrics & Gynaecology
Vice Chairman – ICOG
Vice President, FOGSI (2008-09)
Chairperson-Young Talent Promotion Committee, FOGSI (2003-2007)

POSTMENOPAUSAL OSTEOPOROSIS

Osteoporosis by definition is a disease characterized by low bone mass with microarchitectural deterioration of bone tissue leading to enhanced bone fragility and susceptibility to fracture.

INTRODUCTION

It is a common ailment seen in postmenopausal women, resulting in fragile and weak bones highly susceptible to fractures of hips, spine, and wrist. One in three women over age 50 years will develop the disease during their lifetime. Loss of 20% bone mass in 5 to 7 Prevalence years following menopause is seen. Osteoporosis can be fatal and more women die of hip fractures, than from cancer of ovaries, cervix, and uterus combined. It is a silent disease, because bone loss occurs without symptoms.

One estimate suggests that 65 million people in India suffer from osteoporosis. Incidence of osteopenia is 37% in males and 41.6% in females. Osteoporosis is 5.5% in males and 14.2% in females.

<table>
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<tr>
<th>Prevalence</th>
<th>Persons with osteoporosis (millions)</th>
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<tbody>
<tr>
<td>2003</td>
<td>2013</td>
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<tr>
<td>Females 30%</td>
<td>21.3</td>
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<tr>
<td>Male 6%</td>
<td>4.6</td>
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<tr>
<td>Total (millions)</td>
<td>25.9</td>
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PREVENTION: Role of gynaecologist:
At Adolescent & Adult age: To achieve peak bone mass.
Perimenopause: To prevent osteoporosis in the high risk group
Late Postmenopausal period: To prevent age related osteoporosis.

Improving calcium nutrition, vitamin D intake, regular exercise program, hormonal replacement therapy is very essential and should be started in time, calcitonin to prevent further fractures, no smoking and moderate alcohol intake.

For postmenopausal woman calcium intake of 1 gram per day appears to be necessary to effect a positive impact of exercise on bone mineral density in spine. Study show that intermittent cyclical treatment with etidronate disodium (HEBP) and calcium plus alphacalcidol may be effective for increasing BMD and preventing fractures in postmenopausal osteoporosis.

Treatment:

Treating of postmenopausal osteoporosis should be made after considering patients age, BMD results, history of previous fractures, high risk factor for bone loss.

In conservative management high doses of calcium intake with exercise program would be continued without any osteoporotic drug and to repeat a BMD test after 2 years. If evidence of bone loss is seen on repeat test, antiresorptive medication should be considered. Some patients with keen personal health beliefs, who are very much concerned about skeletal status, and wanting to stay active and fracture free, take and prefer medical therapy at initial stage only. It keeps them anxiety free and allows participation in all activities. Either plan is appropriate depending on quality of life related issues, objectives and concerns.

Treatment options of Osteoporosis - Drug therapy used to treat osteoporosis may be divided into 2 categories: antiresorptive and anabolic agents.
Antiresorptive agents inhibit bone resorption. Anabolic agents stimulate bone growth and formation.

The antiresorptive agents consist of 7 U.S. Food and Drug Administration (FDA)-approved
medications for the treatment of osteoporosis: the bisphosphonates (alendronate, risedronate, and ibandronate), the selective estrogen receptor modulator (SERM) raloxifene, estrogen, and calcitonin. There is currently 1 FDA-approved anabolic agent, teriparatide.12,18,28

1. Antiresorptive Agents
   - Estrogen / Raloxifen
   - Calcitonin
   - Bisphosphonates (Alendronate)

2. Bone forming agents
   - Vitamin D metabolites eg. (Calcitriol, Alpha-calcidol)

3. Adjuvant therapy
   - Calcium
   - Bone building micronutrients like P, Mn, Cu, Zn, etc

It is easier to prevent osteoporosis than to treat it. A good option is to delay menopause associated bone loss.

1. Antiresorptive agents

   The mechanism of action of antiresorptive agents involves a decrease in osteoclastic bone resorption, with no effect on osteoblast function and, hence, the development of new bone. These agents decrease the rate of the remodeling process and reduce the number and severity of resorption sites. The increase in BMD as a result of this therapy is due to the ability of bone formation to occur at a quicker rate than bone resorption due to the presence of such agents.

1. Hormones / Estrogens:

   The role of long term postmenopausal HT in the prevention and management of osteoporosis remains controversial following publication of the results of the Women’s Health Initiative (WHI) study of combined oestrogen and prog-estin therapy (cHT) and its study of oestrogen alone therapy (ET).30,31 The WHI study initiated these HT in women aged 50–79 years, many of whom had cardiovascular risk factors. Women with known osteoporosis were excluded, but the WHI population was otherwise not screened for osteoporosis risk (unlike the bis-phosphonate and raloxifene studies). Despite this, significant reductions in subsequent osteoporosis fractures were seen in both arms of the trial. With so many endpoints, there is controversy about the appropriate adjusted confidence intervals; but the one risk that clearly reached statistical significance was a doubling of thromboembolism. Overall, between the ages of 50 and 79 the annual increase in stroke was around one per 1000 women treated, but the absolute risk was low in the 50s and increased with age. A trend to an increased risk in cardiovascular disease was seen only in the cHT arm of the WHI and was significantly raised only in those initiating cHT over 70 years of age.
Antiresorptive Agents Prevent or Treat Osteoporosis

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In the cHT arm of the WHI, an increase in breast cancer was seen by 5 years of eight per 10 000 (<0.1%) per year. This was matched by a similar reduction in other major cancers. In the WHI study there were no changes in overall cancer and mortality rates. There was a different risk profile to combined therapy for women without a uterus and on this oestrogen only regimen.

A long term trial of HT from early menopause would be required to see if there is any primary neuro- or cardio-protective effect; but following the WHI cannot be recommended for these indications. The risks of HT do appear to be greater when initiated in later age, especially if there is established arterial disease. However, to date the data from randomised controlled trials, observational, animal and laboratory studies suggest a better risk/benefit profile when HT is used from around the time of menopause. Thus, HT is an option for the prevention of osteoporotic fractures particularly in the at risk symptomatic woman around early menopause.

Ideally, oestrogen therapy should be continuous (ie. without a break in therapy). Adjuvant progestogens are necessary in women who still have a uterus to protect against endometrial cancer. They may be given cyclically for 10-14 days each month in perimenopausal women or as continuous therapy combined with oestrogen in postmenopausal women. The latter is more suitable for women more than 2 years postmenopause to avoid the initial irregular bleeding normally seen with this regimen being unduly prolonged.

The optimal dose of HT required to prevent bone loss may vary from woman to woman. With any therapy for osteoporosis, repeat bone density should be considered after 2 years to check if there is improvement.

Despite its advantages, 75% discontinuation rate is seen after 6 months. Recommended dosage is 0.625 milligram Premarin (conjugate estrogen) shown to preserve bone.

Estrogen hormone therapy is FDA approved for the prevention of osteoporosis associated with menopause. Estrogen blocks cytokine signaling the osteoclast, which decreases bone resorption. Results of the Women’s Health Initiative (WHI) revealed that after 5 years of hormone therapy using Prempro (conjugated equine estrogens/medroxyprogesterone), the risk of vertebral fractures and hip fractures was reduced by 34%. This trial randomized 16,608 healthy postmenopausal women between the ages of 50 and 79 with an intact uterus, to receive conjugated equine estrogen-medroxyprogesterone or placebo for 8.5 years. The primary endpoint was an evaluation of cardiovascular events. This was established to examine the relationship between estrogen therapy and cardiovascular complications. The primary adverse endpoint was the frequency of breast cancer. Secondary endpoints included fractures, VTE, stroke, and colon and endometrial cancer. This trial was halted after 5 years due to an increase in participant adverse affects. The relative increase in risk was 41% for stroke, 29% for cardiovascular disease (CVD) events, and 26% for invasive breast cancer.

The ability of estrogen to increase BMD has been demonstrated in women of early and late menopausal phases. The administration of low-dose (0.3-0.45 mg) conjugated equine
Antiresorptive Agents Prevent or Treat Osteoporosis

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Estrogen in combination with calcium and vitamin D produces an increase in BMD and a reduction in bone resorption. Results of WHI have led to concerns regarding an increased risk of cardiovascular events and breast cancer, which have caused a decline in the use of estrogen as an optimal therapy for osteoporosis prevention.

**False perceptions about HRT:**

HRT be used for bone protection because of its unfavourable safety profile, but these perceptions are due to old data, incomplete analysis of the data. Some officials recommend that HRT to be used only in symptomatic patients, or as a second line of management when other medications failed, or contraindicated.

**Recent Evidence:**

States that HRT is effective in osteoporosis related fracture. No evidence suggests superiority of bisphosphonates or any other antiresorptive agents over HRT.

It is a cost effective 1st line treatment.

2. **Tibolone**

It is an alternative to estrogen therapy and its effect on BMD appears to be similar. There are currently no antifracture data available, but an ongoing randomised controlled trial (LIFT) has vertebral fracture as a primary outcome. It is unclear whether the concerns raised about other endpoints from the WHI apply to tibolone.

3. **Calcitonin (Miacalcin; Novartis), a polypeptide hormone derived from salmon, partially inhibits osteoclast activity and has FDA approval for the treatment of osteoporosis in women who are >5 years past menopause. It is available as a daily intranasal spray (200 IU) or subcutaneous injection.**

One of the clinical applications of this product is the reduction of pain associated with acute compression fractures. The mechanism for analgesia is unexplained. Side effects include nausea and nasal congestion. Adverse effects associated with nasal administration include dryness, itching, and epistaxis. Injectable administration may cause local reactions, flushing, and rashes.

4. **Bisphosphonates:**

Non hormonal drug used to inhibit osteoclast activity so decreasing bone resorption, with no adverse effect on breast and uterus and no venous thromboembolism. The oral bisphosphonates are the most widely prescribed agents of the antiresorptive therapies and are considered first-line therapy for the treatment of postmenopausal osteoporosis. The currently marketed products are second- and third-generation bisphosphonates. Etidronate and
Antiresorptive Agents Prevent or Treat Osteoporosis

clodronate are first-generation bisphosphonates that are not approved for treatment of osteoporosis in the United States but are available in Europe. The bisphosphonates (alendronate sodium, risedronate sodium, and ibandronate sodium) are structural analogs of pyrophosphate, a naturally occurring inhibitor of bone resorption. Using a targeting-carrier system, the bisphosphonate prodrug rapidly delivers the compound to the surface of the bone. These agents bind hydroxyapatite crystals of the bone with high affinity and inhibit bone resorption by decreasing osteoclast activity and growth. After the inhibition of resorption, these agents may be eliminated renally or become affixed to the bone matrix, where they reside until remodeling begins again. It is this mechanism that is responsible for the long half-lives of these agents.

Two oral bisphosphonates have been approved to prevent postmenopausal osteoporosis.

1. Alendronate (Fosamax)
2. Risedronate.

They reduce the risk of single, multiple and morphometric (asympto-matic) vertebral fractures in women with osteoporosis and one or more baseline vertebral fractures.16–19

Alendronate is approved by the FDA for prevention and treatment of osteoporosis in postmenopausal women. It reduces the incidence of hip, spine, and wrist fractures by 50% over a 3-year period in patients with a previous spine fracture. Alendronate reduces the risk of spine fractures by 48% over a 3-year period in patients with no history of a spine fracture. The dosing of alendronate for these indications is 5 mg daily or 35 mg weekly for prevention of fractures and 10 mg daily or 70 mg weekly (with or without 2800 IU of vitamin D3) for treatment of fractures. This product is available as a 70-mg once-weekly oral solution. The alendronate tablet should be administered with a full glass of water (6–8 oz) after waking. Neither formulation should be taken at bedtime. Patients should be instructed to remain upright and refrain from ingestion of other foods or medications for at least 30 minutes after medication consumption. Alendronate is not metabolized systemically. It is not recommended for patients with severe renal insufficiency. 6, 33

The Fracture Intervention Trial (FIT) examined the effect of alendronate on the rate of vertebral and nonvertebral fractures in postmenopausal women. 34 This trial was randomized, double-blinded, placebo-controlled, and took place at 11 community-based centers. Women ages 54–81 with femoral neck BMD of 0.68 g/cm2 or less but no vertebral fractures were enrolled. The total enrollment was 4432 women, with 4272 completing the study. Subjects received calcium and vitamin D supplementation during the study period and were assigned to placebo or 5 mg of alendronate for the initial 2 years, then 10 mg of alendronate daily or placebo for the final 2 years. Results determined that alendronate increased BMD at all sites (p < .001) and reduced clinical fractures from 312 in the placebo group to 272 in the treatment group, but results were not statistically significant (14% reduction; 95% confidence interval [CI] 0.73–1.01). The rate of clinically identified fractures was decreased by 36% in women with osteoporosis at the femoral neck (>2.5 SDs below normal young adult mean; 95% CI,
0.50–0.82; number needed to treat [NNT] 15), but there was no significant reduction among patients with high BMD. Alendronate decreased the rate of vertebral fractures by 44% overall (RR, 0.56; 95% CI, 0.39–0.80; NNT, 60), but it did not increase the risk of adverse effects compared with placebo.

Alendronate increased BMD in patients with initially low BMD of −2.5 or less. Patients with higher BMD levels were unaffected by 4 years of therapy. There was an overall reduction in the risk of clinical fractures among women with osteoporosis and with hip or spine T-scores of −2 or more.

Alendronate & risedronate also reduced the risk of vertebral fractures by 50% in women who have osteoporosis without a pre-existing vertebral fracture.17–18 The risk reduction with potent bisphosphonates is usually seen within the first 6–12 months. Peripheral fracture rates are also reduced with alendronate and risedronate in patients with a prevalent vertebral fracture. Data for antihip fracture efficacy are also available. In the alendronate trials there was consistency in hip fracture risk reduction, but hip fracture was not a primary end point.17,20 In one risedronate trial in which hip fractures were the primary endpoint, there was a 40% reduction in hip fracture risk among women aged 70–79 years with osteoporosis confirmed on DEXA (baseline t-score <-3).21 In this study, women aged 80 years and over who had largely been included because of fall risk factors rather than low BMD did not receive the same benefit.

The use of alendronate & risedronate has been associated with dyspepsia, abdominal pain & oesophageal ulceration and should be prescribed with caution in patients with a history of reflux oesophagitis or hiatus hernia.22 However, the overall risk of gastrointestinal events with alendronate and risedronate is very low and weekly bisphosphonates appear to further reduce the risk of this side effect.

**Risedronate**

It reduces the risk of spine fractures up to 49% and non-spine fractures by 36% over a 3-year period in patients with a prior spine fracture. The manufacturer recommended dosing for risedronate is 5 mg daily or 35 mg weekly for both prevention and treatment of bone fractures. Risedronate is absorbed in the upper GI tract and reaches maximum absorption within 60 minutes. Serum steady-state levels are achieved in approximately 56 days. Risedronate is not systemically metabolized. There is no dosage adjustment necessary for patients with renal insufficiency.6,35

**Etidronate**

It is used in a cyclical regimen for osteoporosis, usually for 2 weeks every 3 months because it can result in mineralisation defects if used continuously. A number of smaller controlled trials with etidronate show increases in lumbar spine bone density averaging 5% over 2–3 years23 and suggest a 50% reduction in vertebral fracture rate. Etidronate has been associated with lower, but not upper, gastrointestinal events. There appears to be no risk of mineralisation defect with the cyclical regimen.
Ibandronate

It is the newest agent in this class, a once-monthly tablet of 150 mg, or 2.5 mg daily, approved by the FDA for prevention and treatment of postmenopausal osteoporosis. Ibandronate may reduce the risk of fractures by 50%. The absorption of ibandronate occurs in the upper GI tract. The oral bioavailability is reduced by 90% when administered concomitantly with a standard breakfast. This product is not metabolized in humans. Approximately 50%–60% of the dose is not absorbed by the bone and is eliminated via renal excretion. Ibandronate is not recommended in patients with severe renal impairment.

Bisphosphonates are polar (water soluble) drugs and when taken orally the bioavailability is low (<1%). Calcium should not be taken at the same time of day as a bisphosphonate as it interferes with their absorption. Bisphosphonates should also be taken at least 30 minutes before meals to allow adequate absorption. In patients who are intolerant of oral bisphosphonates, intravenous bisphosphonates such as pamidronate and (more recently) zolendronate are sometimes used. The increase in bone mineral density that occurs with prolonged use of bisphosphonates (>5 years) is maintained for 2–3 years after cessation.24 This does not occur with hormone therapy, where BMD losses commence soon after stopping the drug.25

These drugs have shown to increase vertebral and hip BMD, and decrease vertebral fracture risk. Daily dose of 5 mg per day or once weekly is as effective as daily dosage formulation.

Pharmacokinetics of bisphosphonates:

The administration of oral bisphosphonates requires that patients take them after waking with 8 oz of plain water before any other food or drink of the day and remain sitting upright for 30 minutes (60 minutes if ibandronate is administered). Sitting upright reduces the potential of a possible esophageal injury. Patients should also be instructed to not eat or drink anything for at least 30 minutes after ingesting the drug. The most common side effect is GI upset.

Discontinuance of bisphosphonates: if patients experience symptoms of esophageal disease or injury, musculoskeletal pain, eye inflammation and jaw necrosis.

There are a number of published case reports (>2000) describing jaw necrosis associated with bisphosphonate therapy. Woo and colleagues conducted a recent systematic review to evaluate the incidence of jaw necrosis in patients receive Among 368 published reports, the researchers discovered 94% of cases occurred in patients with metastatic carcinoma to the skeleton or multiple myeloma who were taking intravenous bisphosphonates (zolendronic acid, pamidronate)
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Current FDA recommendations suggest that cancer patients receiving chemotherapy or corticosteroids and those with poor oral hygiene receive a dental examination before initiating bisphosphonate therapy. Recommendations from the American Dental Association also suggest that the incidence of bone necrosis associated with oral bisphosphonate therapy is low (0.7 cases per 100,000 person-years' exposure). Patients should receive a comprehensive dental examination before the initiation of oral therapy. Conservative dental surgical procedures should be used, if needed. 40

Contraindications

Patients with hypersensitivity to the components of the drug, renal insufficiency, hypocalcemia, or esophageal irritation.

Intravenous pamidronate has been used to treat osteoporosis as off-label use. It is an option for women that cannot tolerate or absorb oral bisphosphonates. A loading dose of 90 mg is usually administered, followed by 30 mg every 3 months. The efficacy of pamidronate in the reduction of fractures has not been established. 18

Intravenous zoledronic acid is approved for the treatment of malignant hypercalcemia and multiple myeloma and has the ability to increase BMD and suppress bone resorption in postmenopausal women for 1 year after a single 4-mg dose. The safety and efficacy of this therapy for osteoporosis is being evaluated by the FDA. 12, 41

d) SERMS (selective estrogen receptor modulator)

Selective estrogen receptor modulator raloxifene slows bone decomposition by blocking cytokine signaling to the osteoclast. Raloxifene has both agonist and antagonistic activity exhibited by its ability to interact in an agonistic fashion with estrogen receptors in the bone while interacting antagonistically in the breast and uterus. Raloxifene is FDA approved for the prevention and treatment of postmenopausal osteoporosis, at a dose of 60 mg daily. This product decreases vertebral fractures by 40% in as early as 1 year while increasing BMD. Raloxifene reduces the risk of spine fractures by 30% in patients with and by 55% in patients without a spine fracture over a 3-year period. The risk of breast cancer and coronary heart disease in patients taking raloxifene is under investigation. Clinicians should be aware of an increased risk of venous thromboembolism (VTE) present with raloxifene administration. The most common adverse effects of raloxifene include hot flashes and leg cramps. Raloxifene is contraindicated in patients with a history of VTE. 12, 41
Raloxifene is a selective oestrogen receptor modulator (SERM) which acts to decrease bone resorption like oestrogen but without stimulating the breast or uterus. Lipid profiles are improved and breast cancer incidence has been reported to be reduced by 60–70% over 4 years. Controlled clinical trials with raloxifene have shown modest increase in bone density, although this is generally somewhat less than that seen with bisphosphonates or oestrogen. In women with prevalent vertebral fractures, a 36% reduction in vertebral fractures was noted using a dose of 60 mg per day for 4 years. In women without prevalent vertebral fractures, the relevant risk reduction was greater (55%).

Nonvertebral fractures were not reduced in the main trial analysis for reasons that are unclear. However, as a group, the patients were generally younger (mean age 66.5 years) than in bisphosphonate studies, and hip fracture reduction was not considered as an endpoint in the MORE study. In a posthoc analysis of the MORE study, raloxifene resulted in a significant 14% reduction in peripheral fractures in a sub-group of women who had radiographical evidence of severe spinal deformities (>40% compression). An increased risk of deep venous thrombosis has been reported with raloxifene users similar to that seen with hormone therapy (HT) users.

Treatment should be stopped if patients are immobilised for any prolonged period. Unlike HT, raloxifene is not useful for the control of (and may worsen) menopausal symptoms. Raloxifene has also been shown to be effective for prevention of postmenopausal bone loss and should be considered as an alternative in women unable to take oestrogen for this indication.

It does not ease menopause symptoms, but may exacerbate hot flushes. Clinical trial information to assess the use of raloxifene in postmenopausal women for the treatment of osteoporosis is supplied by the MORE (Multiple Outcomes of Raloxifene Evaluation) trial. Results of this trial indicate that over a 36-month period, administration of raloxifene (60 mg daily) reduced the risk of vertebral fractures by 30% (relative risk [RR], 0.7; 95% CI, 0.5–0.8), reduced the frequency in both men and women without prevalent fractures, and increased BMD at all studied sites ($p < .001$). The risk of nonvertebral fractures was not reduced significantly compared with placebo with the administration of raloxifene.

**Calcium**

Calcium is weakly antiresorptive and supplementation may reduce negative calcium balance, particularly in older age. Most studies suggest the required daily intake is between 1000 mg and 1500 mg in postmenopausal women not taking oestrogen replacement therapy. Controlled trials have found small effects of calcium supplementation on bone density averaging 1–2%
associated with a modest reduction in fracture risk in some studies.33,34

In patients using bisphosphonates, calcium must not be taken at the same time of day as the bisphosphonate or the calcium will impair absorption of the drug. Calcium carbonate may also cause mild constipation or upper gastrointestinal upset. Calcium supplements should be avoided in patients with a history of renal calculi in the presence of hypercalcinuria. Patients presenting with moderate to severe vitamin D deficiency (serum 25 hydroxy-vitamin D <30 nmol/L) are at high risk for osteomalacia and hip fractures and require long term high dosage vitamin D supplementation (up to Ostelin 2000–5000 IU/day). The role of supplemental vitamin D for preventing fractures has been demonstrated in a number of studies. In one French study involving vitamin D deficient institutionalised elderly patients, simple vitamin D3 (800 IU) and calcium (1200 mg/day) reduced hip fractures by 43%.36

Calcitriol (active vitamin D metabolite) has a therapeutic profile distinct from vitamin D. The evidence on efficacy in fracture prevention is confusing, with studies showing both increased and deceased numbers of fractures. Calcitriol should not be used as a sole therapy for the treatment of osteoporosis.

Parathyroid hormone (PTH) stimulates osteoclasts and osteoblasts in the bone, but when presented to bone intermittently such as in daily subcutaneous administration, it has a net anabolic action. It increases cancellous bone mass by about 15–20% over 3 years and reduces the relative risk of vertebral fractures by up to 65% in women with osteoporosis and one or more baseline fractures as well as peripheral fractures. In view of its cost, it is anticipated that PTH will become a treatment option for individuals with severe osteoporosis with ongoing fractures who have failed other therapies.38

Strontium ranelate has been shown in clinical trials to reduce the risk of both vertebral and peripheral fractures. In the 3 year TROPOS study, a 41% reduction in vertebral fractures (relative risk 0.59; 95% confidence interval, 0.48–0.73) associated with an increase in lumbar spine and femoral neck BMD occurred.39 The exact mechanism of action of strontium is unclear, but an antiresorptive action has been described.

MONITORING:
Patient education is essential for treatment adherence. Bone densitometry using DEXA measurements can be used for monitoring the efficacy of therapy because of their precision; they can be performed rapidly and conveniently. It should be noted that changes of less than 5% are within the measurement error of most machines and therefore should be regarded as representing no significant change. It is often recommended that a repeat DEXA be performed within 1 year of starting osteoporosis treatment, especially in the case of corticosteroid induced osteoporosis. For other patients, measurement at 2 years is likely to reflect more accurately the effect of an antiresorptive drug. Biochemical measures of bone turnover may become useful in the management of the individual patient, but their role has yet to be established.

Summary

Osteoporosis is preventable and treatable. Proper use of available therapies by clinicians may enhance the lives of patients, thereby improving their quality of life. Early detection and administration of essential vitamins and supplements, in addition to proper diet and exercise, may greatly decrease possible catastrophic events that seem to debilitate so many postmenopausal women. The use of oral bisphosphonates as first-line antiresorptive therapy is both effective and safe when used in combination with calcium and vitamin D. The appropriateness of the bisphosphonate of choice is a clinical decision that may be individualized per patient, according to the patient's current clinical status, such as renal function or swallowing capability, and other confounding factors (patient compliance, concurrent medication regimen). Calcium and vitamin D supplementation are critical to bone formation and bone remodeling homeostasis. A number of products are available, including tablets, liquid, and soft chewable forms, which should aid in administration efforts. Anabolic pharmacologic intervention for patients with severe bone loss has proven to be extremely promising.

CONCLUSION

ERT considered good choice for women who need therapy for other postmenopausal symptoms. Raloxifene considered for women who do not have troublesome vasomotor symptoms and want a medication that improves BMD without risk to breast and uterus. Bisphosphonates are excellent for women who need to prevent bone loss only.

Barring about drug selection, clinicians should recommend adequate intake of calcium.
to 1500 mg per day) and vitamin D (400 to 600 IU per day) and resistant type exercises to augment the drug benefit. Interventions to Stave off bone loss can help to improve postmenopausal women quality of life and avoid serious consequences of fracture. Clinicians should take lead in prevention strategies that can increase BMD and decrease fracture risk. "Medical experts agree that osteoporosis is highly preventable".

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SUMMARY

Osteoporosis is preventable and treatable. Proper use of available therapies by clinicians may enhance the lives of patients, thereby improving their quality of life. Early detection and administration of essential vitamins and supplements, in addition to proper diet and exercise, may greatly decrease possible catastrophic events that seem to debilitate so many postmenopausal women. The use of oral bisphosphonates as first-line antiresorptive therapy is both effective and safe when used in combination with calcium and vitamin D. The appropriateness of the bisphosphonate of choice is a clinical decision that may be individualized per patient, according to the patient's current clinical status, such as renal function or swallowing capability, and other confounding factors (patient compliance, concurrent medication regimen). Calcium and vitamin D supplementation are critical to bone formation and bone remodeling homeostasis. A number of products are available, including tablets, liquid, and soft chewable forms, which should aid in administration efforts. Anabolic pharmacologic intervention for patients with severe bone loss has proven to be extremely promising.

Clinicians must be vigilant in encouraging patients to be screened and treated as per guidelines. Such actions will decrease overall healthcare costs and improve the long-term quality of life of our patients.