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Guidelines for the management of postmenopausal osteoporosis for GPs

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BACKGROUND

Since the last series of guidelines on the management of osteoporosis from Osteoporosis Australia was published in Australian Family Physician (October 2002), there have been further advances in our understanding of the treatment involved in both the prevention of bone loss and the management of established osteoporosis.

OBJECTIVE

This article provides updated guidelines for the management of postmenopausal osteoporosis to assist general practitioners identify those women at risk, and reviews current treatment strategies.

DISCUSSION

Osteoporosis and its associated problems are major health concerns in Australia, especially with an aging population. While important principles of management are still considered to be maximising peak bone mass and preventing postmenopausal bone loss, new clinical trial data about drugs such as the bisphosphonates, raloxifene and oestrogen have recently become available and the relative role of various agents is gradually becoming clearer. The use of long term hormone therapy has mixed risks and benefits that requires individual patient counselling.

Osteoporosis is defined as a compromise in bone strength, related to a decrease in bone mass/density and an alteration in bone quality (architecture, microcrack and microdamage accumulation, mineralisation, bone turnover and collagen quality) resulting in osteofragility fractures.[1] Clinically, osteoporosis is synonymous with low bone density. An osteoporotic fracture is one that occurs with minimal or no trauma, typically defined as a fall from standing height or less.

In 2001, approximately 2 million Australians were estimated to be affected by osteoporosis, three-quarters of whom were women. This condition is more prevalent than hyperlipidaemia and incurs more years of healthy life lost than Parkinson disease, cervical cancer or rheumatoid arthritis. The total cost burden of osteoporosis was estimated at $7.4 billion per year.[2] The health burden of osteoporosis will only increase as our population ages.[3] The risk of developing osteoporosis is increased by certain factors (*Table 1*) and by some medical conditions (*Table 2*).

Diagnosis

History and examination

A relevant history should include age of menarche, age of menopause, smoking history, prior fracture, family history of osteoporosis and medical conditions listed in *Table 2*. Routine examination of postmenopausal women should include measurement of height, with a 2 cm height decrease over 3 years suspicious for osteoporosis.[4] Assessment for kyphosis may also be used, although in many elderly women this spinal deformity may occur due to severe spondylosis or scoliosis.[5]

Investigations

Dual energy X-ray absorptiometry (DEXA) remains the gold standard for the diagnosis of osteoporosis. Other investigations such as plain X-ray and blood tests may assist with diagnosis, management and follow up.

DEXA

All postmenopausal women sustaining a low trauma fracture should be considered for bone densitometry because of the high likelihood of having osteopaenia/osteoporosis.

Dual energy X-ray absorptiometry measures a patient's bone mineral density (BMD) to help predict fracture risk. It has a reported precision (reproducibility) in research studies of about 1.0–1.5% at the spine and about 3% at the proximal femur. In day-to-day practice compared with research studies precision may be somewhat less (see *Monitoring therapy*). Postmenopausal women lose bone at a rate of approximately 1–2% per year, therefore biennial scans are generally adequate. In patients who are likely to have increased bone loss (eg. glucocorticoid treatment), more frequent scans may be indicated.[6]

Bone mineral density is reported both as an absolute value and in terms of a 't-score', which represents the number of standard deviations (SD) from the young normal mean BMD (*Table 3*). For every one SD decrease in BMD (ie. a t-score reduction by one), the relative risk of fracture approximately doubles.[7] Currently, total hip BMD is most commonly used to predict overall fracture risk, as proximal femoral measurements are less affected by osteoarthritis which can falsely elevate spine BMD values. The z-score is the number of standard deviations from population mean for age (ie. age matched). As t-scores can vary between different types of DEXA machines, the Australian and New Zealand Bone and Mineral Society is attempting to standardise reporting by use of the Geelong Osteoporosis Study reference population.[8] Doctors should encourage DEXA providers to report their results using this reference population.

Table 1. Factors associated with increased risk of osteoporosis

- Increasing age
- Menopause (especially premature)
- Family history of osteoporosis
- Previous low trauma fracture
- Medical conditions (Table 2)
- Low calcium intake
- Low body weight (defined as BMI <20)
- Eating disorders associated with decreased weight
- Immobilisation
- Lifestyle factors including smoking, alcohol, lack of exercise or excessive exercise

Table 2. Medical conditions associated with increased risk of osteoporosis

- Prolonged glucocorticoid therapy
- Conditions associated with excess glucocorticoid secretion
- Amenorrhoea lasting more than 6 months before the age of 45 years
- Primary hyperparathyroidism
- Chronic liver disease
- Chronic renal disease
- Malabsorption (eg. coeliac disease)
- Rheumatoid arthritis and other inflammatory arthropathies (eg. ankylosing spondylitis)
- Conditions associated with thyroxine excess

NB: A Medicare rebate is available for bone densitometry in these situations

In older women, fracture risk is dependent on both age and BMD. That is, a woman 70 years of age with a BMD t-score value of –2.5 or less may have the same risk of fracture as a woman 50–60 years of age with a BMD t-score value of –3 to –4.

Plain X-ray

Plain films are used commonly to diagnose peripheral fractures, but also play an important role in the diagnosis of clinical (ie. painful) and asymptomatic (morphometric) vertebral fractures. A loss of greater than 20% of vertebral height is diagnostic of a vertebral fracture and in the absence of a history of trauma, suggests osteoporosis. In women over 65 years of age, spinal X-ray becomes important when normal bone densitometry may be misinterpreted as a result of spondylosis, vertebral compression or extra-skeletal calcification.

Blood tests

Blood tests are usually normal, but may be indicated to exclude other specific medical conditions (*Table 2*). This is particularly important where the z-score is <–1.5 on bone densitometry. Typical investigations might include:

- full blood count (FBC)
- erythrocyte sedimentation rate (ESR)
- calcium
- creatinine
- total alkaline phosphatase and albumin
- thyroid stimulating hormone (TSH)
- protein electrophoresis (EPP)
- antitissue transglutaminase antibody (TTG) or anti-endomysial antibody
- parathyroid hormone (PTH)
- 25 hydroxy vitamin D.

Biochemical markers of bone remodelling

Serial biochemical markers of bone turnover can be measured in serum and urine and may provide additional information for assessing fracture risk. In longitudinal studies, markers of high bone turnover such as serum bone glaprotein (osteocalcin) or urinary crosslinks/telopeptides have been shown to predict a similar fracture risk as reductions in BMD measured by DEXA.[9] In clinical practice, increased bone turnover markers in the presence of a low BMD would favour treatment of the patient. Additionally, markers may be used to assess the response to treatment and thus increase therapy adherence.[10] However, in clinical practice at primary care level they are rarely needed.

Management of postmenopausal osteoporosis and osteopaenia

Although osteoporosis is a common condition, a cause should be identified if possible and the specific disease processes treated appropriately. Decisions to commence therapy should take account of the patient's age and t-score. In general, interventions are indicated for women with bone densities more than 2.5 SD units below the young normal mean (t-score <–2.5), with preventive measures to be considered before that level is reached. Accordingly, the following intervention guidelines are suggested (*Figure 1*):

- consider preventive measures in postmenopausal women with significant osteopaenia, ie. t-scores between –2.0 and –2.5 as osteoporosis is likely with time. As mild to moderate osteopaenia (t-score between –1.0 and –2.0) in women aged 50–60 years will have a relatively low absolute risk of fracture, it is more difficult to justify treatment for potentially large numbers of women in this category, and
- strongly recommend treatment in postmenopausal women with osteoporosis, ie. BMD values lower than 2.5 SD below the young normal mean (t-score <–2.5) and in patients with osteoporotic fractures.

Normal	= t-score >–1
Osteopaenia	= t-score between –1 and –2.5
Osteoporosis	= t-score <2.5

NB: WHO criteria apply to lumbar spine, hip and forearm only

Table 3. WHO definitions of low bone density for postmenopausal women using DEXA

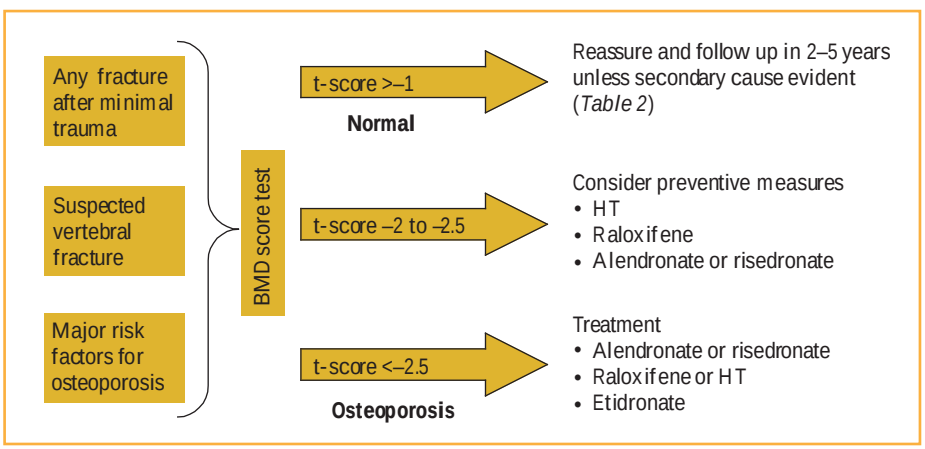

Figure 1. Intervention guidelines
NB: There are no data that intervention in women with mild to moderate osteopaenia (t-score –1.0–2.0) in the absence of a prevalent fracture is beneficial in reducing the risk of subsequent osteoporotic fractures. Therapy aimed at prevention of bone loss may be warranted in some clinical situations. The imperative to consider intervention increases as the t-score decreases

General lifestyle measures

Recommendations apply to both optimisation of bone strength as well as minimising fall risk. These include lifestyle modification such as a diet adequate in calories, protein, calcium, and vitamin D, elimination of smoking and excessive alcohol intake, and exercise and fall prevention measures which may be beneficial and prevent fractures. The optimal use of exercise relies upon delivery of a sustained adequate dose of the correct modality of exercise to the target population while minimising the risk of side effects.[11]

Medication

Although lifestyle prevention measures are important, pharmaceutical agents may be recommended for the prevention of postmenopausal bone loss. Randomised trials have shown beneficial effects of potent bis-

phosphonates, selective oestrogen receptor modulators, oestrogen and tibolone in prevention of postmenopausal bone loss.[12–15] As most of the controlled trials of antiresorptive agents described below have used concomitant calcium and vitamin D, it is appropriate to add a calcium supplement to most therapies except calcitriol.

Bisphosphonates

Bisphosphonates are potent inhibitors of bone resorption. Currently three bisphosphonates are approved in Australia on the Pharmaceutical Benefits Scheme (PBS) (Authority required) for the treatment of established osteoporosis in postmenopausal women with fracture due to minimal trauma. Alendronate and risedronate reduce the risk of single, multiple and morphometric (asymptomatic) vertebral fractures in women with osteoporosis and one or more baseline vertebral fractures.[16–19] Alendronate and risedronate also reduce 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 endpoint.[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. This highlights the need to also address falls related factors in women of advanced years.

The use of alendronate and risedronate has been associated with dyspepsia, abdominal pain and 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.

Etidronate 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 years[23] 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.

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) zoledronate are sometimes used. Further studies assessing antifracture efficacy with these regimens are in progress. 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]

Selective oestrogen receptor modulators

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.[26] 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%).[27] 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 subgroup of women who had radiographical evidence of severe spinal deformities (>40% compression).[28] An increased risk of deep venous thrombosis has been reported with raloxifene users similar to that seen with hormone therapy (HT) users.[26] 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 is available on the PBS (Authority required) for established postmenopausal osteoporosis in patients with fracture due to minimal trauma. 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 (no PBS listing for this purpose).[29]

Hormone therapy

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 progestin 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 bisphosphonate and raloxifene studies). Despite this, significant reductions in subsequent osteoporosis fractures were seen in both arms of the trial. There were trends to other mixed risks and benefits which differed between the two therapeutic regimens. 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. The absolute increased risk of this morbidity varied with age and other thrombotic risk factors from one in 10 000 in a healthy woman aged 50 years up to one in 100 in a woman aged 80 years. 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.

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 (*Figure 2*). In the WHI study there were no changes in overall cancer and mortality rates.[30] In 2004, the ET arm of the WHI study ceased after 6.8 years showing a reduction in breast cancer of seven per 10 000 per year (*Figure 3*).[31]

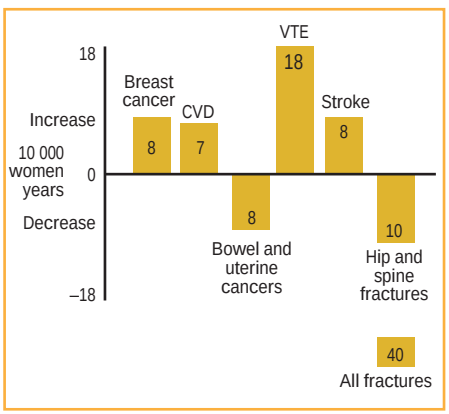

Figure 2. Annual disease risk after 5 years of combined HT[30]

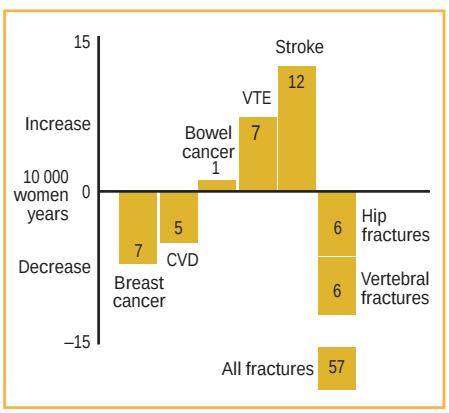

Figure 3. Annual disease risk after 7 years of oestrogen only HT[31]

This suggests a different risk profile to combined therapy for women without a uterus and on this oestrogen only regimen. Again the absolute risk for vascular disease rose with age.

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.

As with all therapies, women should be fully informed of the risks and benefits of their particular HT regimen, be reviewed annually, and therapy should be individualised. 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 the therapeutic regimen is efficacious.

Tibolone

Tibolone is an alternative to oestrogen therapy and its effect on BMD appears to be similar.[32] 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.

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. This can be obtained from about three serves of dairy products per day. For example, one glass (250 mL) of milk, two slices (40 g) of cheddar cheese, or one tub (200 g) of yoghurt each contains about 300 mg of calcium. For those who are unable to tolerate dairy products, calcium enriched soy milk and a large variety of calcium supplements are currently available from pharmacies and health food stores. However, patients need to understand the varying calcium content of different supplements and that the recommended daily intake relates to elemental calcium. 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 citrate is now available in Australia as an option for these patients. Calcium supplements should be avoided in patients with a history of renal calculi in the presence of hypercalcinuria.

Vitamin D

Vitamin D undergoes metabolism in the liver and kidney. Simple vitamin D is mainly available in Australia as ergocalciferol (Ostelin 1000 IU per capsule) or cod liver oil tablets (approximately 400 IU). Smaller amounts of vitamin D are also contained in some calcium and vitamin supplements (eg. Caltrate + D contains 200 IU per tablet). These preparations are beneficial for preventing vitamin D deficiency in individuals with a poor diet or limited sunlight exposure. However, patients presenting with moderate to severe vitamin D deficiency (serum 25 hydroxyvitamin 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). In Australia, these population groups include the housebound elderly, older women in residential care facilities, dark skinned or veiled women, and individuals with malabsorption.[35]

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] In a recent British study involving elderly community dwelling men and women, cholecalciferol 100 000 units administered orally every 4 months for 5 years resulted in a 35% reduction in all major osteoporotic fractures.[37]

Calcitriol

Calcitriol (active vitamin D metabolite) has a therapeutic profile distinct from vitamin D and should not be used in the treatment of vitamin D deficiency.[6] Calcitriol is available on the PBS (Authority required) for the treatment of postmenopausal osteoporosis in Australia. 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

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. It is now registered in Australia as an 18 month therapeutic course but as yet is not PBS listed. 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

Table 4. When to consider referral to a specialist

- Problems or side effects with treatment
- Other complex medical conditions
- No access to appropriate bone densitometry
- Inadequate response to therapy
- A vertebral fracture (a one visit assessment by a specialist is prudent)
- Young patient (age <50) with osteoporosis
- Secondary cause is identified
- Continued fractures with 'normal' bone densitometry

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. This agent is currently unavailable in Australia.

Thiazide

Several large, prospective epidemiological studies in elderly men and women have shown that thiazide use is associated with a reduced risk of hip fracture. Several possible mechanisms could explain this association. Thiazides act directly on the distal nephron to enhance calcium reabsorption leading to positive calcium balance. Thiazides may also reduce osteoclastic activity possibly by inhibiting carbonic anhydrase. One randomised study of treatment of systolic hypertension included bone mass as a secondary endpoint in a subset of patients. The subjects who took thiazide had increased bone mass whereas those on other forms of antihypertensive medication showed a decrease in bone mass.[40] The effect of thiazides on bone density in patients with normal blood pressure has not been studied, but a randomised trial is underway.

Combination therapy

While there are published data demonstrating an additive effect of combination therapies on BMD, there are no published data regarding additional antifracture benefit and the role of combination therapy is unclear.[41]

Monitoring therapy

Patient education is essential for treatment adherence. This includes information on their disease as well as the importance of intervention therapies. This should be tailored to address individual patient concerns about the specific risks of their treatment.

After therapy has been initiated, it is important to monitor patients to ensure that bone loss is controlled. 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. Thereafter, biennial DEXA assessments are usually carried out to monitor response to therapy. Repeat measurements should generally be performed on the same machine for monitoring, assuming the DEXA provider has good quality control with acceptable precision error. Biochemical measures of bone turnover may become useful in the management of the individual patient, but their role has yet to be established. In certain cases, specialist referral may be able to fine tune therapy (*Table 4*).

Prevention of fragility fractures

Preventing falls

Interventions to reduce the risk of falling include:

- modifying the environment to reduce the risk of slipping and tripping by eliminating slippery surfaces, loose rugs, narrow passageways, dangerous furniture. Most people report trips, slips and loss of balance as the cause of the fall, whereas only a small proportion report dizziness or feeling faint
- modifying living habits:
  - by wearing appropriate footwear
  - taking care when walking up or down steps, especially if wearing bifocals
  - taking care at night and in poorly lit conditions
- installing appropriate aids (eg. supportive handrests, rails and nonslip bathmats)
- modifying medications (eg. sedatives, antidepressants or certain antihypertensives that might predispose patients to falls)
- correcting poor vision, and
- involving community agencies to:
  - provide support services to help imple ment the modifications required to reduce the risk of injury
  - ensuring nursing and physiotherapy services are provided when needed.

Specific exercise programs that emphasise ground reaction forces, muscle strengthening and balance retraining (eg. tai chi, resistance training or high impact exercise) may also be effective. Aerobic training, water exercises, and flexibility/calesthenics have not been shown to be effective forms of exercise for falls prevention.[42,43]

Preventing osteoporosis

Although medical intervention may be required in some individuals, patients should understand that it is important to:

- maximise peak bone density in childhood and adolescence, and
- maintain bone density throughout adult life.

Osteoporosis prevention programs should:

- promote a diet with adequate calcium and advocate regular weight bearing high impact, and/or strengthening exercise in children and adolescents
- exercise in premenopausal women should include resistance training to improve muscle mass, strength and balance, as well as regular weight bearing high impact, and/or strengthening exercises
- exercise in postmenopausal women should include resistance training to improve muscle mass, strength and balance, as well as specific balance training which should be performed three times per week[6]
- encourage good general nutrition and ensure vitamin D status is maintained, especially in elderly patients, and
- discourage tobacco use.

Conclusion

The management of postmenopausal osteoporosis should be based on an individual risk/benefit analysis, time since menopause, presence or absence of oestrogen withdrawal symptoms, history of atraumatic fractures, and other medical conditions. Oestrogen therapy remains appropriate for women at the time of menopause for those with symptoms, and for those with premature or surgical menopause. The duration of use should be reviewed at 2–5 years.

Raloxifene and bisphosphonates are alternatives for prevention of bone loss after menopause when symptoms of oestrogen withdrawal are no longer a consideration; although their use in this fashion before a fracture is currently not subsidised by the PBS. In older women, especially those with a prior osteoporotic fracture and high risk of further fracture, bisphosphonates should be considered as first line therapy because they reduce the risk of both nonvertebral and vertebral fractures. Vitamin D deficiency should be excluded before initiation of treatment. All women should have an adequate calcium intake of at least 1000 mg per day. Simple vitamin D (with calcium) supplementation should be considered in those groups at risk of vitamin D deficiency and osteomalacia, either as monotherapy or in combination with other treatments.

Summary of important points

- The cause of osteoporosis should be identified and specific disease processes should be treated appropriately.
- Whereas primary prevention of fracture remains crucial, treatment to ensure that further fractures do not occur is equally as important.
- Any patient aged 45 years and older presenting with a low trauma fracture should undergo bone densitometry (Medicare rebate available for these patients).
- Women at high risk of osteoporosis (*Table 1, 2*) should undergo DEXA for diagnosis (*Table 2* attracts a Medicare rebate).
- The role of long term oestrogen as first line therapy to prevent bone loss at the time of menopause is controversial. The potential benefits must be rationally considered and discussed with patients balanced against the possible small increase in the risk of breast cancer.
- In older patients with prior fractures, first line treatments are alendronate or risedronate. Raloxifene is an alternative first line treatment to prevent spinal fractures.
- Simple vitamin D supplementation should be considered in the housebound or institutionalised elderly.
- Adequate dietary calcium and a lifestyle that includes the correct modality of exercise and falls prevention are also important.

Acknowledgment

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Resources

Osteoporosis Australia (www.osteoporosis.org.au) has a range of education materials available for patients and health care professionals covering different aspects of osteoporosis and its management. There are regular courses on osteoporosis and self management teaching people how to maintain bone mass, how to avoid falls and fractures, and how to live more comfortably with osteoporosis. Helpline 1800 242 141.

Australian and New Zealand Bone and Mineral Society (www.anzbms.org.au).

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