Menopause and osteoporosis: Non-pharmacological and pharmacological management

Celyanda Goeltom, Shoshana Sztal-Mazer

Background
Once osteoporosis has been identified, risk stratification is followed by non-pharmacological and, where needed, pharmacological management, which may include menopausal hormone therapy (MHT) where indicated.

Objective
This article will build on general practitioners' skills in stratifying fracture risk, supporting their patients to maintain bone health and preventing osteoporotic fracture by instigating lifestyle changes along with appropriate pharmacotherapy.

Discussion
Risk stratification entails integrating fracture history, bone density and FRAX risk into The Royal Australian College of General Practitioners' official modified flowchart. Non-pharmacological treatments, such as ensuring adequate calcium intake and vitamin D sufficiency, form the basis of bone health maintenance. MHT should be considered in women aged <65 years with T-scores between −1.8 and −2.5. Pharmacotherapy is integral to fracture prevention in osteoporosis. Those at very high fracture risk may need a referral to a consultant physician for consideration of bone anabolic therapy.

GIVEN THE MORBIDITY AND MORTALITY associated with osteoporosis,[1,2] treatment is crucial. Risk stratification is indicated, followed by a holistic approach using non-pharmacotherapy and, where needed, pharmacotherapy including menopausal hormonal therapy (MHT) to prevent fractures.

Stratifying fracture risk to guide management

Fracture risk can be stratified from low to very high, although the specific criteria differ across international guidelines. The category of very high fracture risk was introduced to identify patients who may benefit from earlier use of potent anabolic therapies such as romosozumab or abaloparatide as first-line treatment to reduce imminent fracture risk.[3] In Australia, this refers to those with a T-score ≤−2.5 with a symptomatic fracture due to minimal trauma and at least one hip or symptomatic vertebral fracture in the previous 24 months; or at least two fractures including one symptomatic new fracture in the previous 24 months.[3,4] Such patients merit bone specialist referral for consideration of Pharmaceutical Benefits Scheme (PBS)-subsidised first-line therapy with a bone anabolic agent.

All those sustaining hip and/or vertebral fractures require pharmacotherapy with one of the options listed in Figure 1 and elaborated in Table 2, whereby the choice of agent depends on factors such as previous therapy, patient preference and, if available, T-score.[3,5]

In general, any minimal trauma fracture (excluding hands, face and feet) warrants osteoporosis treatment.[3,6] Where possible, dual-energy X-ray absorptiometry (DXA) is helpful to facilitate this; however, this is not necessary from a Medicare Benefits Schedule or good practice point of view. When the T-score is >−1.5 and the minimal trauma fracture was not in the hip or vertebra, consider further investigations (for secondary osteoporosis or malignancy).[5,6]

Those at high risk who have not yet sustained a fragility fracture can be risk stratified with the FRAX tool.[7] In general, a 10-year fracture risk ≥20% for major osteoporotic fracture (MOF) or ≥3% for hip fracture is defined as meeting the treatment threshold. Figure 2 illustrates the MOF risk in a traffic light format to guide management decisions, and it highlights those women for whom anabolic therapy should be considered.

Management

Osteoporosis management requires attention to modifiable lifestyle factors (Table 1) alongside appropriate pharmacotherapy, including MHT.

Vitamin D levels and calcium intake should be optimised before initiating therapy (Table 1). Participants in most trials for osteoporosis pharmacotherapy were vitamin D sufficient and were required to take calcium supplementation.[5,8–10] Guidelines now advise vitamin D and calcium sufficiency prior to osteoporosis pharmacotherapy initiation. If needed, supplementation may be given to achieve this.[6] Renal function should be reviewed to ensure safe prescribing.

When selecting pharmacological treatment, efficacy, contraindications, adverse effects, cost, treatment duration, sequencing and post-treatment consolidation strategies need consideration (Table 2).

There is growing evidence that starting with bone anabolic therapy (romosozumab, teriparatide or abaloparatide) and then transitioning to antiresorptive treatment produces greater gains in bone density than prescribing these therapies in the reverse order, which appears to blunt the bone density response. At the time of writing, only romosozumab is on the PBS for first-line anabolic therapy, although abaloparatide was approved by the Pharmaceutical Benefits Advisory Committee in August 2025 for this indication. This limits choices of first-line anabolic therapy available in Australia, unless patients are able to self-fund teriparatide.[4,11] Of note, all three anabolic agents have second-line indications whereby romosozumab and teriparatide are on the PBS for this indication. Teriparatide is approved by the Therapeutic Goods Administration for up to 24 months, whereas the PBS restricts subsidised use to 18 months.

MHT prevents bone loss and fractures in all postmenopausal women regardless of bone density. This is why some experts and guidelines advocate for MHT in all postmenopausal women with osteopenia (where there are no contraindications).

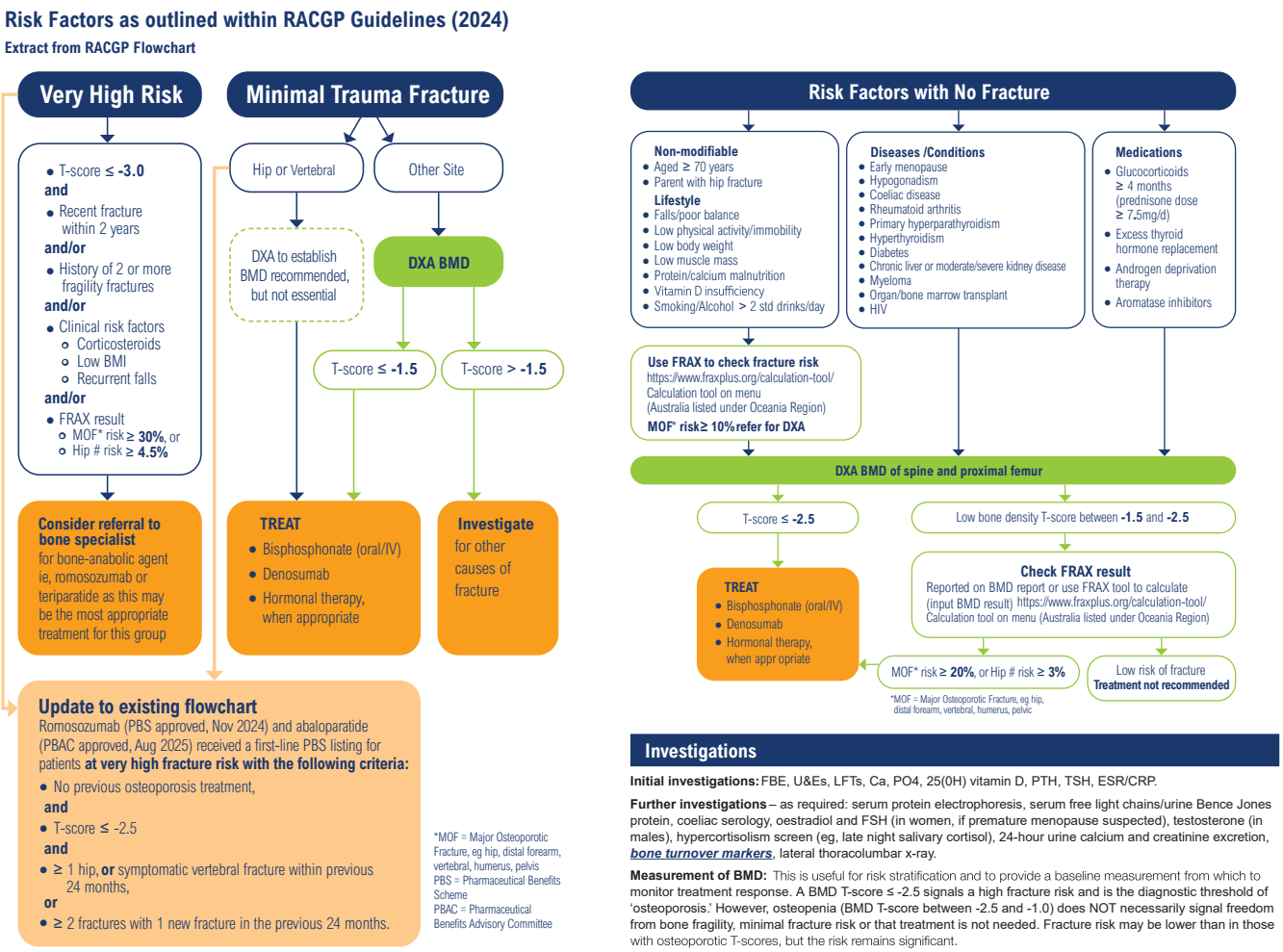


Figure 1. Osteoporosis risk assessment, diagnosis and management flow chart.

Reproduced from Wong P, Chen W, Chiang C, et al. Position statement on the management of osteoporosis. Health Bones Australia, 2025, with permission from Healthy Bones Australia.

Table 1. Non-pharmacological measures to maintain healthy bone

Non-pharmacological treatment	Recommendation	Further detail
Calcium	Recommended dietary intake = 1300 mg/day for women aged ≥50 years.[22]	Calcium is a vital mineral that provides structural strength in the bone matrix. Dairy products are rich in calcium and are better absorbed than non-dairy sources; 3–4 serves/day are recommended. Other calcium-rich foods are tinned fish and tofu. Dietary calcium is better absorbed than calcium supplements. Excessive supplementation may cause renal calculi and gastrointestinal side effects, and there is a possible association with arterial calcification;[23] thus no more than 600 mg supplementation (usually one tablet) is advised, and only if dietary calcium intake is inadequate.[24]
Vitamin D	Target level is 50–100 nmol/L. Levels above 125 nmol/L may be detrimental.[25]	Vitamin D promotes the absorption of calcium and facilitates bone mineralisation.[25,26] In Australia, 20.6% of adults have inadequate vitamin D3.[27] The main source of vitamin D is skin exposure to ultraviolet (UV) B sun rays. Duration and extent of exposure needed depend on the season, location, skin type and area of skin exposed. It is better to expose a large area for a shorter time than a smaller area for longer time.[28] Darker skin poses less risk for skin cancer and does not absorb vitamin D as well, so less sun protection is required, and more sun exposure is needed.[28] It is important to find a balance between sun exposure for bone health and sun protection for skin cancer.[28] Cancer Council recommends sunscreen when the UV rating is ≥3: routinely in those with pale skin, but only with extended outdoor exposure in those with dark skin.[28] Patients with vitamin D <50 nmol/L should supplement with 1000–4000 IU/day depending on the serum level. Recheck 3 months later. If in target (50–100 nmol/L), supplementation should be maintained at 1000 IU/day.[5]
Protein	Recommended dietary intake = 1–1.5 g/kg body weight/day for older adults.[29,30]	Collagen forms the organic matrix of bone, and low protein intake is associated with low bone density.[11,31] Protein is also important for muscle health and increases intestinal calcium absorption.[29] In those who are institutionalised, older adults and those with sarcopenia, dietary protein is particularly important for falls and fracture reduction.[31,32] Dairy foods are an excellent source of readily absorbable protein.
Exercise	A regular combination of weight-bearing, resistance and balance exercises is recommended.[33]	Exercise improves muscle mass and bone density while preventing falls.[34] Gait speed or the Timed Up and Go test are recommended as screening tools for gait and balance programs. The former measures the time taken for older persons to get up from a chair without using their arms, walk for 3 m, turn around and return to their seat to sit down.[34] Prescribe >3 hours of exercise/week of: • 2–3 sets of 5–8 repetitions of progressive resistance (strength) training 2–3 days/week • 50 impacts of 2–4 × body weight ≥3 days/week (eg jumping) • ≥3 sessions of progressive and challenging balance activities.[3,19]
Smoking cessation		Smoking causes microarchitectural damage, meaning that the fracture risk it incurs is greater than that shown by bone density alone. Even passive smoking can have an adverse effect on bone mass.[35] Current smoking increases any fracture risk by 25%, vertebral fracture risk by 80% and hip fracture risk by 40–84%.[31] When smokers stop smoking, the fracture risk declines[32] such that each year of non-smoking reduces the relative risk of fracture by 1%.[34]
Alcohol reduction	Limit alcohol to <3 standard drinks per day (<10/week; <4 on any one day). Even less alcohol may be desirable in terms of other health outcomes.	Regular alcohol consumption of ≥3 drinks/day increases the risk of osteoporosis and fracture.[36] Falls risk is also increased. These risks are independent of bone density.[37] Lowering intake to standard levels reduces fracture risk with, two-thirds of those at high risk of major osteoporotic fracture and 41% of those at high risk of hip fracture moving out of the high-risk category.[38]
Falls avoidance[6]		Falls increase fracture risk. Exercise as above and appropriate vitamin D replacement may lower falls risk. To further lower falls risk: • educate patients on fall reduction measures: loose rugs and wires, appropriate footwear etc. In high-risk cases, home safety assessments and home modification interventions are indicated. • review medication to minimise postural hypotension and sedation with antihypertensives and psychotropics, respectively. • encourage eye checks to ensure safe vision.

Recent updates to the Australian-authored international menopause tool kit recommend MHT to treat osteopenia in women aged under 65 years, regardless of menopausal symptoms, where T-score is ≤–1.8.[12] Some sources suggest that the FRAX tool lacks validity in women aged <65 years, in part because menopausal status and use of MHT are not included in the FRAX scoring system. The basis for the ≤–1.8 cut-off originates from the National Osteoporosis Risk Assessment (NORA) study, which found that women with a DXA result in this range had a short-term increased risk of fracture.[13]

Those with osteoporosis (T-score ≤–2.5) are still advised to be managed according to the national osteoporosis guidelines (Figure 1).

Potential uncommon antiresorptive therapy complications

All bisphosphonates (alendronate, risedronate and zoledronic acid) and denosumab can cause the following rare side effects.

Osteonecrosis of the jaw

The risk of osteonecrosis of the jaw (ONJ)[14] developing depends on:

- patient risk factors, such as duration of osteoporosis therapy (>4 years increases risk) and comorbidities such as diabetes, rheumatoid arthritis and renal impairment[15]
- type of dental work, where a simple extraction and simple implants are low risk, but extensive oral surgery and implants or extractions in the posterior mandible pose a high risk.[16]

Hence, the incidence varies between 0.02% and 0.3% in those on antiresorptive therapy for osteoporosis, but this can increase to 2.5% with invasive dental treatment. Thus, a dental check is recommended before starting antiresorptive therapy; however, treatment to prevent fractures should not be delayed unless, in the opinion of the treating doctor or dental surgeon, the individual has a particularly high risk of developing ONJ.[17]

In general, withholding antiresorptive treatment does not reduce the risk of ONJ, especially when the patient has been on this treatment long term (>4 years). Thus, there is no clear indication to stop bisphosphonate therapy, especially where the dental work is low risk. As denosumab must not be stopped except under the guidance of a bone physician, it is advisable for patients on denosumab who require dental work to have this done at the time of least antiresorptive action. This is approximately 4 weeks before their next dose is due. Those at high risk of ONJ should be referred for dental and consultant physician bone specialist care.

Atypical femoral fracture

Atypical femoral fracture (AFF) risk increases with antiresorptive treatment duration and patient factors such as genetics, Asian ethnicity[18] and comorbidities such as rheumatoid arthritis.[19] Out of all antiresorptive agents, the oral bisphosphonates pose the highest risk. The absolute incidence is 3.2–50 cases per 100,000 person-years of bisphosphonate treatment,[20] with this rate reducing substantially in those who pause their therapy (Figure 3).[19]

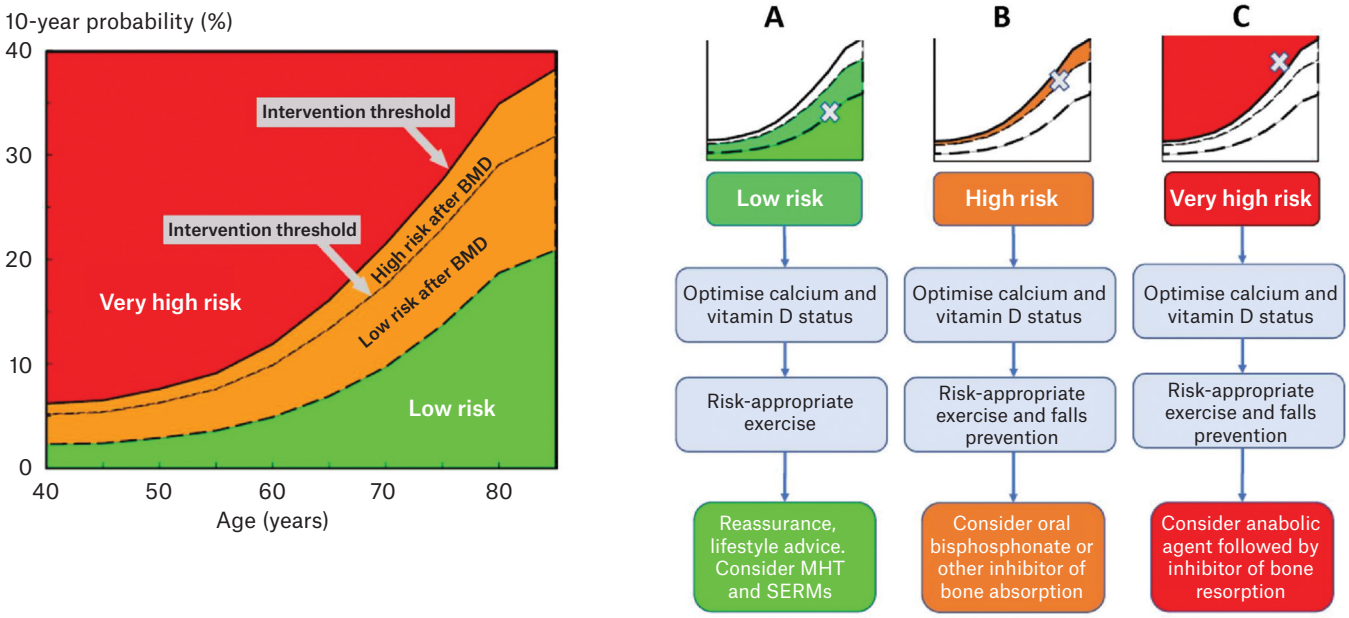


Figure 2. Fracture risk stratification using FRAX 10-year fracture risk calculator.

BMD, bone mineral density; MHT, menopausal hormone therapy; SERM, selective oestrogen receptor modulator.

Adapted from Curtis EM, Reginster JY, Al-Daghri N, et al. Management of patients at very high risk of osteoporotic fractures through sequential treatments. Aging Clin Exp Res 2022;34(4):695–714. doi: 10.1007/s40520-022-02100-4, licensed under a Creative Commons Attribution 4.0 International License.

Patients on long-term antiresorptive agents who complain of upper thigh or groin pain should have bilateral complete femoral plain X-rays to exclude early signs of AFF.[21]

Bone turnover markers

Bone turnover markers are typically used to assess treatment adherence, evaluate therapeutic response and guide recommencement of paused bisphosphonate therapy. Therefore, they can be ordered 3 months after treatment commencement, whenever treatment efficacy or adherence is questioned and every 1–2 years when bisphosphonate therapy is paused.

The bone turnover markers most commonly used in Australia are:

- C-terminal telopeptide (CTX) – a breakdown product of type I collagen that reflects bone resorption. Optimal suppression is generally considered to be <200 mcg/L. CTX must be measured in the fasting state, as food intake can falsely lower results and lead to misinterpretation.
- procollagen type 1 N-terminal propeptide (PINP) – a marker of collagen formation that reflects bone formation. It is particularly useful for monitoring the response to anabolic therapy such as teriparatide, with levels typically reassessed approximately 3 months after treatment initiation.

Conclusion

Non-pharmacological measures – including adequate dietary protein; calcium and vitamin D optimisation; regular weight-bearing, strength-based exercise; and falls prevention – are fundamental aspects of osteoporosis management. Women who have a very high fracture risk may benefit from consultant physician review for consideration of a PBS-listed anabolic agent as first-line therapy. Where first-line anabolic therapy is not indicated, but pharmacotherapy is, antiresorptive therapy with bisphosphonates or denosumab (where adherence and lifelong treatment are not management barriers)

Table 2. Pharmacological management options

Name	Dose	Practice points
Romosozumab	Monthly subcutaneous injections (in two syringes) for 12 months given by the general practitioner. First script must be initiated by a consultant physician.	• Potent bone anabolic agent that blocks sclerostin, an inhibitor of bone formation. It also has antiresorptive action after the first month of therapy. • First-line treatment for very high-risk osteoporosis. • Used as second-line treatment when T-score ≤–3 and ≥2 minimal trauma fractures (MTFs), one of which occurred after ≥12 months continuous antiresorptive therapy. • Reduces vertebral, nonvertebral and hip fractures.[39] • Contraindicated in patients with previous acute myocardial infarction or stroke. • Imperative to consolidate with an antiresorptive agent after completion of romosozumab.
Teriparatide	Daily subcutaneous injection for 18 months. First script must be initiated by a consultant physician.	• Bone anabolic agent that stimulates the parathyroid hormone receptor (intermittently such that bone formation predominates, as opposed to continuously as occurs in hyperparathyroidism whereby resorption overrides formation). • Second-line indication when T-score ≤–3 and ≥2 MTFs, one of which occurred after ≥12 months continuous antiresorptive therapy. • Teriparatide is approved by the Therapeutic Goods Administration (TGA) for 24 months, but only 18 months on the Pharmaceutical Benefits Scheme (PBS). • No PBS indication for first-line treatment, although it is a good first-line agent if patients' finances enable this. • Needs refrigeration. • Not recommended to use after denosumab as may be associated with transient bone loss (but can be used off label alongside denosumab). • Reduces vertebral and nonvertebral fractures. • Imperative to consolidate with antiresorptive treatment after completion of teriparatide.
Abaloparatide	Daily subcutaneous injection for 18 months. Must be initiated by a consultant physician.	• Bone anabolic agent that stimulates the parathyroid hormone 1 receptor and, in turn, osteoblasts to build bone. • Approved by the Pharmaceutical Benefits Advisory Committee for first- and second-line treatment for very high-risk osteoporosis but not yet PBS listed at the time of writing. • Does not need refrigeration. • Reduces vertebral, hip and nonvertebral fractures. • Imperative to consolidate with antiresorptive treatment after completion of abaloparatide.

Table 2 continued on the next page

Table 2. Pharmacological management options (cont'd)

Name	Dose	Practice points
Alendronate and risedronate	Oral bisphosphonates	• Poorly absorbed. To avoid gastric side effects, they should be taken on an empty stomach followed by 30 minutes in an upright position (except enteric coated risedronate, which can be given with food). • Both reduce vertebral, hip and nonvertebral fractures. • Generally use as first-line therapy. • Often used for consolidation after a bone anabolic agent or denosumab. • A pause in therapy after 10 years may be indicated when fracture risk reduces (refer to Figure 3). Follow up with dual-energy X-ray absorptiometry (DXA) and bone turnover markers (fasting C-terminal telopeptide) is required.
Zoledronic acid	Intravenous bisphosphonate, delivered via 12–18 monthly infusion for 3–6 years	• New evidence exists that even less frequent treatment is effective.[40] • Reduces vertebral, hip and nonvertebral fractures. • A pause in therapy after 5 years may be indicated when fracture risk reduces (refer to Figure 3). Follow-up with DXA and bone turnover markers is required. • Can be done in a community infusion centre (www.anzbms.org.au/infusion-centres-for-zoledronic-acid.asp).
Denosumab	6-monthly subcutaneous injection; lifelong use	• Monoclonal antibody that inhibits receptor activator of nuclear factor kappa-B ligand (RANKL), reducing osteoclastogenesis and thus bone resorption. • Should only be commenced in those who intend to be adherent with continuous therapy. • Rapid rebound bone loss occurs when stopped, so need to strictly follow treatment interval, preferably not delaying by more than 4–6 weeks.[41] • When treatment stops, a bisphosphonate should be commenced immediately, generally under specialist guidance. • Reduces vertebral, hip and nonvertebral fractures. • Consider checking calcium level day 11 post injection when estimated glomerular filtration rate <30 mL/min as hypocalcaemia can occur.[3,5] If there is a risk of hypocalcaemia, it is the authors' view that calcium supplementation (approximately 1000 mg daily) should be taken for 2 days before until 2 weeks after each dose.[42,43]
Oestrogen Tibolone	Menopausal hormone therapy; oral or transdermal	• Oestrogen works mostly by reducing osteoclast formation, but it also has an effect on osteoblasts. • Reduces vertebral, hip and nonvertebral fractures.[44] • Can be used for prevention of bone loss in younger women (perimenopause and T-score ≤–1.8) or those with increased fracture risk if there are no contraindications.[12] • Should be used in women with premature ovarian insufficiency (menopause before the age of 40 years) if there are no contraindications.[45] • Consider in women with early menopause (aged 40–45 years) to those aged <65 years for prevention of bone loss when T-score ≤–1.8 even when no menopausal symptoms are experienced, if no contraindication.[1] • Beneficial for women who have intolerable menopausal symptoms. • Consider risk–benefit ratio regarding breast cancer, venous thromboembolism (VTE), treatment duration and age of patient.[40,46,47] • Transdermal form does not increase VTE risk.[42] • Topical vaginal oestrogen does not prevent bone loss. • Tibolone is only used post menopause and up to age 70 years because of the increased risk of stroke in those aged >60 years, which increases further in those aged >70 years.[48–50]
Raloxifene	Selective oestrogen receptor modulator	• Oestrogen agonist on bone. • Slightly increases risk of VTE. • Beneficial in postmenopausal woman who cannot take oestrogen but detrimental to bone density in premenopausal women.[51] • Reduces vertebral and nonvertebral fractures. • Does not improve vasomotor symptoms and may worsen them.

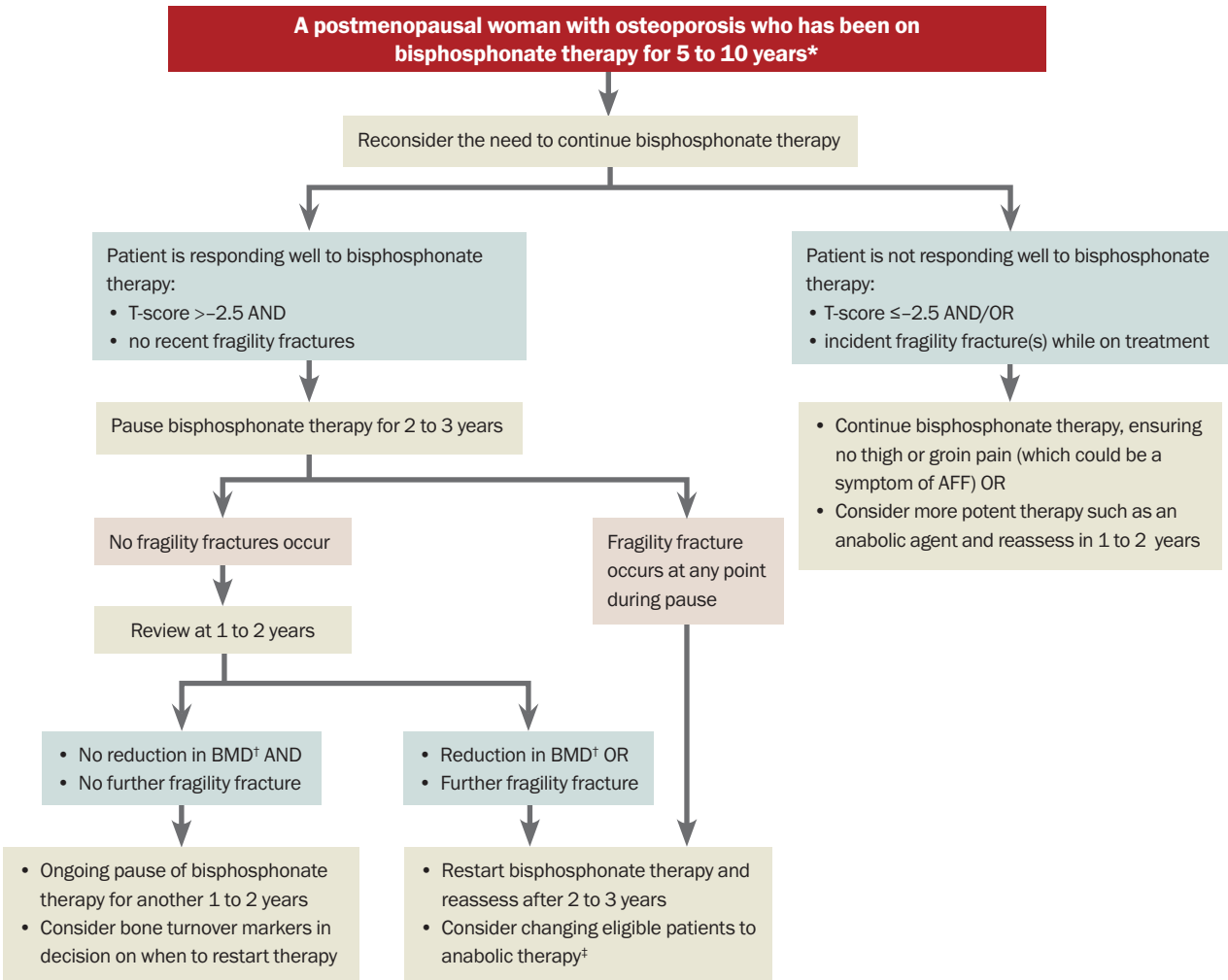
should be commenced. MHT can also be considered in women who have osteopenia where a T-score is ≤–1.8 and there are no contraindications.

The rare adverse effects of ONJ and AFF warrant attention to good dental hygiene and, where appropriate, pauses in bisphosphonate therapy, but undue concern about these adverse effects should not deter appropriate osteoporosis pharmacotherapy.

Key points

- As menopause causes accelerated bone loss, MHT may be a suitable first-line therapy in women aged <65 years with a T-score ≤–1.8.
- It may be appropriate to offer women using long-term bisphosphonates a pause in therapy to reduce the small chance of AFFs.
- Denosumab dosing should not be delayed ≥4 weeks because of rebound bone resorption and increased vertebral fractures risk (ie there should never be a pause in denosumab treatment).
- It may be appropriate to refer women with a high imminent fracture risk to a consultant physician to consider bone anabolic agents first line; or second line when a fracture occurs while on antiresorptive medication.

When to reconsider the need to continue bisphosphonate therapy in postmenopausal women with osteoporosis



Abbreviations: AFF = atypical femoral fracture; BMD = bone mineral density; DXA = dual-energy x-ray absorptiometry.

*This algorithm is also applicable to men but not to those taking denosumab because denosumab therapy cannot be ceased once commenced.

†Least significant change on DXA: ≥4% at lumbar spine or ≥5% at hip if this is not noted on the report.

‡The patient may qualify for anabolic therapy if the fracture occurred within 1 year of previous zoledronic acid dose and the patient has a T-score ≤–3.

Figure 3. When to reconsider the need to continue bisphosphonate therapy in postmenopausal women with osteoporosis.

Reproduced with permission from Jones AR, Sztal-Mazer S. Postmenopausal osteoporosis: Bridging the treatment gap. Endocrinology Today 2024;13(3):28–35.

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Competing interests: None.

Funding: None.

Provenance and peer review: Commissioned, externally peer reviewed.

AI declaration: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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