Osteoporosis treatment

All patients with osteoporosis should be counseled on weight-bearing exercise, smoking cessation, moderation of alcohol intake, and calcium and vita-min D supplementation. Treatment of osteoporosis is influenced by the patient's fracture risk, the effectiveness of fracture risk reduction, and medication safety. Patients at high risk of fracture should consider treatment with antiresorptive therapy, including bisphosphonates and denosumab. Anabolic agents such as teriparatide, abaloparatide, and romosozumab should be considered for patients at very high risk or with previous vertebral fractures [1)].

Drugs

Despite the considerable progress in drug development, there is still a clear need to improve treatment strategies and develop new pharmaceuticals against various types of osteoporosis [2)]

A variety of drugs are safe and effective in preventing osteoporotic vertebral fractures. Hormone replacement therapy (HRT) and calcitonin only reduced the risk of vertebral fractures during a follow-up of 21-72 month [3)]

Drugs that increase bone formation include:

1. Parathyroid hormone analogs (PTAs): lifetime treatment limit of 2 years because the effectiveness diminishes and the risk of osteosarcoma (in rats) increases. Unlike drugs that reduce resorption, PTAs used intermittently (e.g., once daily) stimulate osteoblasts preferentially over osteoclasts with the result of laying down of new bone. FDA-approved for patients at high risk of fracture who: are postmenopausal women, men with hypogonadal osteoporosis, or either gender with osteoporosis due to systemic glucocorticoids. PTAs have been used off-label to treat patients with osteopenia/osteoporosis prior to spine instrumentation. Prior to treatment: check serum levels of intact parathyroid hormone (iPTH) and 25(OH)D. Do not use any vitamin D deficiency is corrected. Monitor Ca + + during therapy (transient rise in Ca + + is common, but per- persistent hypercalcemia has been reported)

✖ Contraindication: patients with increased risk of osteosarcoma (e.g., Paget’s disease, unexplained elevation of alkaline phosphatase), prior radiation therapy involving the skeleton, young patients with open epiphyses, patients with hypercalcemia, ± patients on digoxin (these agents may increase calcium levels)

a) Teriparatide (Forteo®, Bonsity®):

b) abaloparatide(TymlosTM):

2. sodium fluoride: 75 mg/d increases bone mass but did not significantly reduce the fracture rate. 25 mg PO BID of a delayed-release formulation (Slow Fluoride®) reduced fracture rate but may make the bone more fragile and could increase the risk of hip fractures. Fluoride increases demand for Ca++,thereforesupplementwith800mg/dCa++ and400IU/dvitaminD.Not recommended for use > 2 yrs Drugs that reduce bone resorption are less effective on the cancellous bone (found mainly in the spine and at the end of long bones26). Improvement in spine bone mineral density accounts for only a

Vertebral bone mass by > 5% and decreases the rate of vertebral fractures by 50%. Also relieves post-meno- pausal symptoms and reduces the risk of CAD. However, use is limited since HRT increases the risk of breast cancer and breast cancer recurrence as well as DVT

2. calcium current recommendation for postmenopausal women: 1,000–1,500 mg/d taken with meals33 3. vitamin D or analogues: promote calcium absorption from the GI tract. Typically administered with calcium therapy (either calcium or vitamin D alone are less effective). Vitamin D 400– 800IU/disusuallysufficient.IfurinaryCa++ remainslow,high-dosevitaminD(50,000IUq7–10 d) may be tried. Since high-dose formulations have been discontinued in the U.S., analogues such as calcifediol (Calderol®) 50 mcg/d or calcitriol (Rocaltrol®) up to 0.25 mcg/d may be tried with Ca++ supplement.Serumlevelsof25-hydroxyvitaminD[25(OH)D],AKAcalcidiolisthebestindi-

small part of the observed reduction in the risk of vertebral fracture.

1. estrogen: cannot be used in men. Estrogen hormone replacement therapy (HRT) increases verte cator of vitamin D status.

4. calcitonin: a hormone synthesized by the thyroid gland which decreases bone resorption by osteoclasts. May be derived from a number of sources, but salmon is one of the more common ones. The skeletal response is maximal during the first 18–24 months of therapy. The benefit of preventing fractures is less well-established

a) parenteral salmon calcitonin (Calcimar®, Miacalcin®): indicated for patients for whom estrogen is contraindicated. Expensive ($1,500–3,000/yr) and must be given IM or sub-Q. 30–60% of patients develop antibodies to the drug which negates its effect. ℞: 0.5 ml (100 U) of calcitonin (given with calcium supplements to prevent hyperparathyroidism) SQ q d

b) intranasal forms(Miacalcin nasal spray):less potent(works better in older women>5yrs post menopause). 200–400 IU/d given in one nostril (alternate nostrils daily) plus Ca + + 500 mg/d and vitamin D

5. bisphosphonates: carbon-substituted analogs of pyrophosphate have a high affinity for bone and inhibit bone resorption by destroying osteoclasts. Not metabolized. Remain bound to the bone for several weeks

1)
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2)
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3)
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