

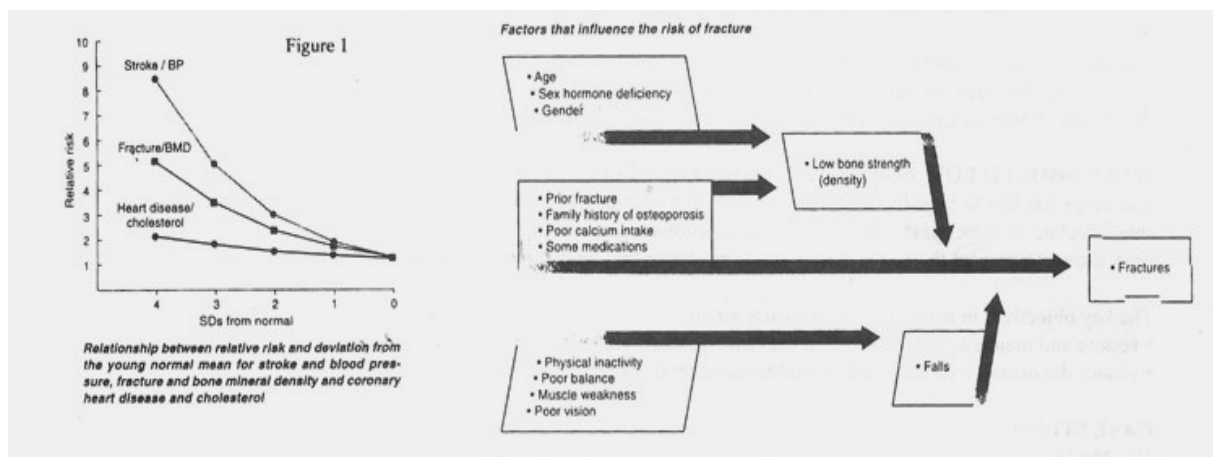
TREATMENT OPTIONS FOR LOW BONE DENSITY: APPENDIX 2

It is estimated that the proportion of women with osteoporosis (T score < -2.50) increases from 15% in those aged 60 to 64 years up to 71% in those over 80 years of age. The frequency is much lower in men, ranging from 1.6% of those 60 to 64 years up to 19% in those over 80 years of age.

BONE DENSITY AND FRACTURE RISK

There is a continuous inverse relationship between bone density and the risk of fracture, comparable to that between serum cholesterol and the risk of coronary heart disease and that between blood pressure and the risk of stroke (Figure 1).

Since bone density measurements can be made at various sites with different machines and at different ages, the values are expressed more usefully in relation to population mean values. Within an age group, one standard deviation (SD) fall in bone density multiplies the relative risk of fracture by 2-3 (see diagram of risk factors below).



THE IMPORTANCE OF THE T SCORE

Patients with a T score between +1 and -1 do not require treatment, although dietary calcium and weight bearing exercise need to be emphasised. Two yearly follow-up bone densitometry assessments are important in peri-menopausal women in case of rapid bone loss. Generally, five yearly follow-ups are sufficient.

Patients with a T score between -1 and -2.5 on bone densitometry have osteopenia. Anti-resorptive treatment to prevent further bone loss should be considered. Medical intervention should have minimal side effects. The presence of risk factors or predisposing conditions will influence the decision to treat.

Life style changes the patient is able to undertake include weight-bearing exercise, adequate calcium intake (supplements with vitamin D may need to be considered) and ceasing smoking. Bone mass needs to be monitored every 1 – 2 years.

Patients with a T score < - 2.5 require specific medical intervention. The concern is to prevent further bone loss and improve bone mass if possible. The need to minimise or prevent falls needs to be discussed. Bone mass needs to be monitored every 1 to 2 years. Independent risk factors such as fractures with minimal trauma or a history of falls will influence the decision to treat a patient.

EFFECTIVE INTERVENTIONS IN PREVENTING OSTEOPOROSIS AND OSTEOPOROTIC FRACTURES

A health life style can be advocated in childhood, adolescence and early adulthood, including regular exercise, adequate calcium intake and avoidance of smoking or excessive alcohol intake (1).

Specific therapies

Calcium – either through sufficient calcium intake in the diet or calcium supplements. A daily intake of 1000-1500mg is needed in all age groups. This is an effective approach in osteopaenic women over the age of 60. However, this only modestly slows bone loss and is less effective than hormone replacement therapy in the perimenopausal woman. Osteoporotic female patients irrespective of age require more specific treatment in most instances.

Vitamin D – deficiency is prevalent in elderly hospitalised or nursing home patients due to lack of sunlight, poor oral intake or poor intestinal absorption. Therapeutic doses of ergocalciferol (500 – 1000U/day) reduce hip and non-vertebral fractures.

Hormone replacement therapy – is an anti-resorptive agent. It prevents postmenopausal bone loss and reduces fracture risk by increasing bone mass. It may also be indicated in women with postmenopausal symptoms. However, its effects are reversed once HRT is stopped. The research carried out on the safety and effectiveness of HRT makes it the preferred treatment for prevention of bone loss in postmenopausal women where osteoporosis or osteopenia is present. There is no evidence that phytoestrogens and progesterone creams prevent bone loss or increase bone mass in women using these products.

Bisphosphonates – are the treatment of choice for postmenopausal women who are osteoporotic and have sustained minimal trauma fractures. There is a significant increase in bone density of the lumbar spine and femoral neck over two years, and all fractures are reduced by approximately 50%. Alendronate and risedronate are preferred to sodium Etidronate (Didrocal) or Pamidronate (ADP) because of their greater efficacy. Tolerance may be an issue. Alendronate can be given weekly.

Raloxifene is a selective estrogen receptor modulator (SERM). This drug does not stimulate the breast and the uterus. It can be used where HRT is contra-indicated because of past breast disease including breast cancer. It increases bone density and reduces fracture risk at the spine but is unproven in preventing hip or peripheral fractures.

Calcitriol – may be useful in milder forms of postmenopausal osteoporosis and may be useful adjunct therapy to HRT. It is indicated in patients with impaired calcium absorption. It should be avoided in treating vitamin D deficiency. While on calcitriol, calcium supplements should be stopped.

WHEN SHOULD LOW BONE DENSITY BE TREATED?

It is never too late to treat low bone mass. Bone loss continues in old age and may accelerate as bone resorption increases. A small decline in bone mass results in a disproportionate rise in bone fragility. Preventing bone loss, even in the elderly, has been shown to prevent the increase in fracture incidence that would occur if treatment had not been given (1).

The key objectives in managing osteoporosis are to:

- Restore and maintain bone strength to prevent fractures; and
- Reduce the overall morbidity and mortality associated with the condition.

CASE STUDIES

1. **Mr M.C.: DOB 02.06.57** – History of long bone fractures and vertebral fractures, in some instances with minimal trauma. High bone turnover on biochemical assessment. No cause found.

Bone mineral density (g/cm ²)	08/96	08/97	09/98
Femoral neck	0.709	0.776	0.752
AP lumbar spine	9.871	0.869	0.909

High calcium intake, alendronate commenced in August 1996. There has been a 6/1% increase in BMD at the femoral neck and a 4.3% increase at the lumbar spine. Response to alendronate has been modest and delayed.

2. **MRS M.W.: DOB 03.08.23** – History of rheumatoid arthritis well controlled with prednisolone 7 mg daily and methotrexate 7.5 mg weekly. BMD demonstrated severe osteoporosis, no vertebral fractures on plain radiology. Initial investigations total serum calcium 2.62mmol/l, ionised calcium 1.2mmol/l PTH 12.31uU/. Diagnosis: primary hyperparathyroidism. Biochemical screening: high bone turnover

Bone mineral density (g/cm ²)	06/96	08/98
Femoral neck	0.632	0.564
AP lumbar spine	0.832	0.710

There has been a 10.8% decrease in bone mass at the femoral neck and a 1.7% decrease at the lumbar spine (neck exploration undertaken but a parathyroid adenoma was not identified). Alendronate 20mg daily has been commenced in the last 6 months. Bone turnover has been suppressed with alendronate, hypercalcaemia persists.

Comment:

Case 1 – whether a response to treatment is occurring can be monitored with DEXA. An alteration in treatment, either type or dose, can be instituted.

Case 2 – In patients rapidly losing bone mass, secondary causes of osteoporosis need to be excluded. The rapidity and severity of bone loss may lead to more active treatment to slow the process.

(1) The 1996 Consensus Conference on Osteoporosis (Med.J. Aust., 167 suppl. 7-Jul, 1997).