For appointments at Albany, Milford, Epsom, Botany and Henderson :
Ph: 09-623-2301
Fax: 09-623-2302
Healthlink: akldbone
Email:
admin@bonedensity.co.nz

NEWSLETTER

Autumn 2007

AUCKLAND Bonedensity
Managing Bone Health

The importance of vitamin D in skeletal health.

Vitamin D plays a critical role in skeletal homeostasis. Sunlight acts on skin precursors to generate pre-vitamin D, which is in turn converted in the liver to the main circulating form of vitamin D, 25-hydroxyvitamin D. 25(OH)D is then further hydroxylated in the kidney to 1,25 dihydroxyvitamin D, which promotes intestinal absorption of calcium and phosphate, and can activate osteoclastic bone resorption.

Severe deficiency of vitamin D leads to impaired bone mineralization (osteomalacia) by causing deficiency of both of the important mineral components of bone. Less severe vitamin D deficiency causes secondary hyperparathyroidism and accelerated bone loss (osteoporosis). Vitamin D deficiency may also cause a proximal myopathy that increases the risk of falls, and therefore fractures.

Values below 50nmol/L should be considered vitamin D insufficient.

Sunlight deprivation causes vitamin D insufficiency. Thus, institutionalized elderly patients, dark-skinned individuals and those who avoid sunlight for cultural or medical reasons are at high risk of vitamin D deficiency. In these groups of patients, routine vitamin D supplementation is recommended. (see below re Calciferol dose).

However, about 2/3 of independent-living older women are vitamin D insufficient during winter months. Thus, many women diagnosed with osteoporosis, for whom anti-resorptive therapy such as bisphosphonates is subsequently prescribed, will have suboptimal vitamin D status.

For this reason, routine prescription of vitamin D supplements to postmenopausal women commencing treatment with a bisphosphonate is sensible.

This is most easily accomplished by administering Calciferol (1.25mg tablet) once monthly.

Steroid Therapy and Fosamax

Potent bisphosphonates (e.g., alendronate/Fosamax) do prevent bone loss in the face of steroid therapy. Bone loss may be otherwise rapid especially in older people.

Your patients on Prednisone (>5mg/day for > 3 months) are eligible for Fosamax if:

1. They have had an X-ray proven fragility fracture (you apply for SA).
   or
2. No fracture history but a DXA T-score is <-1.5 (we will automatically apply for Special Authority if obtained at one of our Facilities).

It seems that many eligible patients are not receiving bone protection. We can help provide you the evidence and expedite the process.

Physicians

Assoc-Prof . Geoff Braatvedt MD, FRACP	Assoc-Prof . Andrew Grey MD, FRACP	Dr Brandon Orr-Walker FRACP	Prof. Ian Reid MD, FRACP
Dr Pat Frengley FRACP, FACP	Prof. Ian Holdaway MD, FRACP	Dr. Warwick Bagg MD, FRACP	Dr. Jenny Lucas MBChB, FRACP

AUCKLAND BONE DENSITY NEWSLETTER

Autumn 2007

AUCKLAND Bonedensity Managing Bone Health

THYROID DISEASE UPDATE

There have been several important advances in thyroid medicine reported in the last year:

- Patients with impaired gastric acid require increased doses of thyroxine. This is particularly relevant in patients on proton pump inhibitors, and dosage review of thyroxine should follow initiation of these medications, utilising TSH monitoring in the usual way. Typical dosage increases are in the order of 30-40%.

- Thyroid autoimmunity in women has been known to be associated with higher rates of miscarriage, and this persists even in euthyroid women. A recent study has shown that thyroxine treatment, initiated at the first ante-natal visit (typically prior to 10 weeks gestation by dates) is associated with lower rates of prematurity and miscarriage to the level seen in thyroid antibody negative women. This study needs to be replicated in another patient group to assess generalisabilty, before we can recommend embarking on this approach.

- Patients often ask whether their thyroxine replacement dose is optimised, and some request dose increases to manage "persisting hypothyroid symptoms". Small changes in thyroxine dose bringing mean TSH levels from the upper half of the normal range (mean 2.8 mU/L) to lower normal, (mean 1.0) or at lower limit (mean 0.30) do not improve any hypothyroid symptoms, or alter quality of life. There is no evidence that the target TSH in hypothyroid patients should deviate from the general laboratory range of normal.

- Is subclinical hyperthyroidism (TSH below reference range with normal free T4 levels) important? Previously, TSH levels below 0.1 mU/L with normal free T4 has been associated with higher rates of atrial fibrillation. This is confirmed in a recently reported large (3000 patients) prospective study (11 years) of people over 65 years. Those with TSH< 0.5 had higher rates of AF (approx 55% vs. 30%, $p < 0.001$). In older patients, TSH below the normal range, if persistent, is associated with adverse cardiovascular risk, as well as accelerated bone loss.

- Is subclinical hypothyroidism (TSH 4-10 with normal Free T4) associated with increased risk of ischaemic heart disease? No increased risk was found in a large (3000 patients) prospective cohort study.

- There was brief interest in using combination thyroxine with tertroxine (T4+ T3) replacement therapy after a single trial showed benefit in some symptoms. Subsequent studies failed to reproduce this effect. A recent meta-analysis of all of the 11 randomised trials comparing T4 with T4 + T3 (1200 patients) has found no added benefit with combination therapy with respect to bodily pain, anxiety, depression, fatigue, quality of life, body weight or cholesterol (and subfraction) levels. There remains nothing to recommend tertroxine coadministration with thyroxine in routine treatment of hypothyroidism.

Refs:

- Centanni et al NEJM 2006;354:1787-95
- Negro et al JCEM 2006;91(7);2587-91
- Walsh JP et al JCEM 2006; 91(7):2624-30
- Cappola et al JAMA 2006; 295(9):1033-41]
- Grozinsky-Glasberg et al JCEM 2006; 91(7):2592–2599