THE INFLUANCE OF HYPOTHYREOSIS AND L-THYROXIN THERAPY ON METABOLIIC BONE TURNOVER

Abstract: The metabolic bone turnover is regulated with many systemic hormones and local growth factors.Additional influences include nutrition (especially calcium)and exercises.

Hyperthyreoidism causes high bone turnover by accelerating activation frequency of bone–remodeling units, leading to decreased cortical and trabecular bone volume.

Thyroid hormones can be stimulate bone resorption directly through thyroid receptors on bone cells. It is now belived that TSH has direct influence on bone mineral remodeling. Even a 50% reduction in TSHR expression produces significant bone loss.

Replacement and mildly suppresive doses of L-thyroxine are not associated with significant bone mineral loss. Patients receiving L-thyroxin replacement therapy with low serum levels of TSH have ,despite lower bone mineral densities than patients with normal TSH,no evidence of an increased fracture rate. Patients,during the first year L-thyroxin replacement therapy of overt hypothyreosis have a significant cortical bone loss.

We must insist on optimal supstitution. In a high-risk group, who need thyroid hormone replacement therapy, the risk of fracture may be prevented with estrogen replacement therapy, adequate calcium supplement, exercise and eventual medical prevention (bisphosphonates antiresorptive therapy) of osteoporosis.

Key words: bone turnover, hypothyreosis, L-thyroxin, TSH.