CLINICAL EFFECTS OF THYROID HORMONES ON BONE

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Abstract

Linear growth and skeletal maturation occur during fetal and childhood development and continue until epiphyseal fusion occurs. This process results from endochondral ossification in the epiphyseal growth plates of long bones and is regulated by systemic hormones as well as paracrine and autocrine factors. The major regulators of development and childhood growth are growth hormone (GH), insulin-like growth factor 1 (IGF-1), glucocorticoids (GC), and thyroid hormones.

The actions of thyroid hormones on bone and their effects on calcium homeostasis result in a negative calcium balance in thyrotoxicosis. There is increased urinary calcium excretion, increased urinary phosphate reabsorption, increased fecal calcium loss, and reduced intestinal calcium and phosphate absorption. Serum albumin concentrations are reduced, resulting in elevated mean ionized calcium and phosphate concentrations. Marked hypercalcemia is uncommon but may occur in up to 5% of subjects; mild hypercalcemia occurs in up to 20%. The relative hypercalcemia suppresses parathyroid hormone (PTH) secretion and serum PTH concentrations correlate negatively with free thyroxine levels. Biochemical markers of bone turnover are elevated in hyperthyroidism and reflect increased activities
of both osteoblasts and osteoclasts. Serum markers of osteoblast-mediated bone formation (alkaline phosphatase and osteocalcin) are elevated and correlate with thyroid hormone concentrations.

Levels of a serum marker of bone collagen synthesis, carboxyterminal propeptide of type I procollagen, are also elevated in thyrotoxicosis and correlate with other bone formation markers, although the relationship with serum T4 concentrations is variable. Urinary markers of osteoclastic bone resorption (pyridinoline and deoxypyridinoline collagen crosslinks and hydroxyproline) are increased in thyrotoxicosis and all three correlate with severity of disease. The serum levels of carboxyterminal cross-linked telopeptide of type I collagen, a specific marker of type I collagen degradation in bone, are also elevated in thyrotoxicosis and correlate with thyroid hormone concentrations and other markers of osteoclast activity.

Thyroid hormones have been recognized in recent years to play an important physiological role in skeletal development, linear growth, and the maintenance of bone mass. Alterations in thyroid status result in growth abnormalities, bone loss, and increased fracture risk.