



Workshop Abstract

W12

Anabolic therapies for osteoporosis

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Therapies for osteoporosis are either anti-catabolic or anabolic. Subcutaneous teriparatide [human PTH (1-34)] injections are the first anabolic therapy for osteoporosis and significantly increases BMD and reduce vertebral and non-vertebral fracture risk. There are no hip fracture data. Its mode of action is differs from anti-catabolic drugs in that it restores trabecular microarchitecture and increases cortical and trabecular bone volume.

Teriparatide increases bone formation before bone resorption, allowing for an early anabolic response. Early increases in type I procollagen propeptides strongly predict the subsequent increase in BMD and bone microarchitecture. However, increases in areal BMD underestimate the effects of teriparatide in increasing bone strength. Results from 3-dimensional micro-computed tomography of biopsies show teriparatide significantly increases trabecular bone volume, connectivity and cortical bone thickness without loss of cortical bone porosity versus placebo. Thus, the increased biomechanical competence may explain the observed reductions of vertebral and non-vertebral fracture incidence of patients treated with teriparatide.

Mild transient hypercalcemia may occur; however, monitoring of serum calcium is not required. Osteosarcoma was observed in a rat oncogenicity study, but no cases of osteosarcoma in response to teriparatide have been reported in human studies and only a handful have been reported associated with hyperparathyroidism. Studies using PTH (1-84) are underway. Combining PTH and anti-catabolic drugs diminishes PTH effects; sequential therapy is preferred. Anti-catabolic therapy should be reinitiated after PTH to maintain or increase BMD.

Another drug, strontium ranelate, reduces vertebral and non-vertebral fracture risk. Although it increases osteoblastic cellular activity, definitive data relating to its effects on bone histomorphometric bone formation are not currently available.