THE EFFECT OF TERIPARATIDE ON BONE REMODELING IS NOT RELATED TO SERUM LEVELS OD OSTEOPROTEGERIN AND RANKL

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In vitro studies indicate that parathyroid hormone (PTH) decrease osteoprotegerin (OPG) secretion by the osteoblasts and increase RANKL production. Studies in healthy men have shown a negative correlation between endogenus PTH and serum OPG; moreover a decrease of serum OPG with an increase of serum RANKL have been reported by an interventional study where postmenopausal women with glucocorticoid osteoporosis were treated with intermittent injections of PTH.

The aim of this study is to evaluate the behaviour of serum OPG and RANKL in women with established postmenopausal osteoporosis treated for six months with teriparatide (20 µg/die).

We enroled 21 women (aged 71.5±7.8 years) with one or more vertebral fractures and a T score at densitometric examination of lumbar spine or proximal femur lower than –2.5. Patients on treatment were included in the study after a three months washout period. As control group, we studied 15 age-matched women. A blood sample was drawn from each patient at baseline and after two, four and six months from the beginning of PTH administration. Bone turnover was evaluated by: specific bone alkaline phosphatase (BSAP), osteocalcin (BGP), and intact N-terminal propeptide of type I procollagen (PINP) as markers of bone formation; C-telopeptides of Type-I collagen (CTX) as markers of bone resorption. Serum OPG and RANKL were assessed using a sandwich enzyme immunoassay (Osteoprotegerin and RANKL, Biomedica, Austria). Precision intra and interassay was lower than 10%. Basal values of bone markers, serum OPG and RANKL in treated patients and control group did not differ significantly. No correlations were found between bone markers and serum OPG and RANKL at baseline. Patients on PTH treatment showed a significant increase in both formation and resorption markers. The increase of BGP and PINP resulted statistically significant after two months, while the increase of CTX became significant after four months. The highest values of all markers were found at sixth months. Serum OPG and RANKL levels did not change significantly, though they showed a tendency to increase. OPG peaked at fourth month, while the highest value of RANKL was found at sixth month. These data confirm that teriparatide stimulates bone formation by activating osteoblast function which leads, later, to osteoclast activation. Serum OPG and RANKL do not appear to be involved in parathyroid action on bone.