Summary

Osteoporosis is a serious social and medical problem. About 6% people suffer from osteoporosis in European countries. As for aetiology it is a multi-factorial disease. Endocrinopaties play an important role in it's development. We focused on three specific endocrinopaties with relation to bone metabolism in our study.

Goal of the study: 1/ To evaluate the influence of long term thyroid hormone therapy (substitution and suppressive dose) on bone mass and bone metabolism.
2/ To evaluate the possibility of prediction of bone mass increase after the operation in patients with mild primary hyperparathyroidism.
3/ To evaluate changes of bone mass, bone metabolism and body composition in patients with growth hormone deficiency treated with growth hormone substitution.

Patients and methods: 1/ a) First - cross sectional part of the study – 76 patients on suppressive dose of L-thyroxine therapy, 34 patients on substitution therapy. We examined bone markers and bone mass in each group. We evaluated how each of the two doses of therapy influences the bone metabolism and we compared the groups with each other. b) In second - prospective part of the study - we observed the changes of bone mass in the course of the time (5 years) in each group.
2/ Retrospective cross-sectional study - 54 patients with primary hyperparathyroidism. We examined the bone markers and bone mass before and one year after the successful operation. We correlated average change of bone density one year after the operation with preoperative parameters.
3/ Prospective study (8 years) – 32 patients with growth hormone insufficiency. We annually evaluated the influence of growth hormone substitution therapy on bone mass and body composition (fat and lean body mass).

Results: 1/ We observed normal bone density in patients on substitution and suppressive L-thyroxine therapy. We did not find out significant differences in bone density of L-spine between groups of patients on suppressive or substitution therapy. The bone density of proximal femur was lower in the suppressive therapy group, the difference was at the border of statistical significance. Bone density was significantly lower in postmenopausal women on suppressive therapy in comparison with women on substitution therapy, predominantly in proximal femur. There was no significant difference in levels of bone markers between the suppressive and substitution groups. 5-year suppressive therapy did not lead to significant decline of bone mass in comparison with substitution therapy.
2/ The increase of bone density in proximal femur and L-spine correlated strongly with bone markers. The correlation with parathormone level was less strong, with serum and urine calcium it was weak or insignificant.
3/ Bone density increased in the period of the growth hormone substitution therapy. Body fat (total or trunk) decreased during the first years, with stable levels afterwards. Lean bone mass increased during the first years, with stable levels afterwards.

Conclusion: 1/ The long-term substitution therapy has no negative influence on bone mass. However, suppressive therapy brings about some risk of bone mass loss, especially in postmenopausal women. 2/ There exist predictors of bone mass increase after operation for primary hyperparathyroidism. 3/ We proved positive and probably long-lasting effect of growth hormone substitution therapy on bone mass in adult patients. We proved positive effect on body composition as well. The period in which this effect will persist is the matter of further studies.