de acção dos fármacos anabólicos e dos fármacos anti-reabsortivos e os estudos farmacológicos e clínicos efectuados apontam para um provável benefício para os doentes em usar ambos os agentes de forma complementar, isto é, iniciar o tratamento com um anabólico e seguidamente fazer um anti-reabsortivo.

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PP03. HYPERTHYROIDISM AND BONE

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Overt hyperthyroidism is a clinical condition caused by exaggerated levels of circulating thyroid hormones. Some of its main etiological factors are the hyperfunction of the thyroid gland and the iatrogenic cause, like the ministration of excessive doses of thyroid hormones. Some studies have shown that the prevalence of hyperthyroidism in women aged 65 or more, varies between 5 and 15%. The potential risks of hyperthyroidism are diverse and can vary from patient to patient; however, heart and bone complications are relatively common, especially among the elderly. Regarding the adult skeleton, several anomalies were described, namely reduced bone mineral density (BMD) and a higher osteoporotic fracture risk. Indeed, hyperthyroidism has been recognized to be an important cause of secondary osteoporosis and a risk factor for hip fracture in women. Moreover, these osteoporotic fractures are associated with a risk of precocious mortality, namely in the elderly. In adult life, after the acquisition of the peak bone mass, the excess of circulating thyroid hormones can lead to an increase in bone resorption, however, the mechanisms involved in their skeletal action are far from totally clarified. While T_3 is considered an important regulator of the bone tissue integrity and of the bone formation, T_4 can stimulate directly or indirectly the activity of osteoclasts. Bone remodeling accelerates while the bone formation period is decreased, originating an incomplete substitution with new bone cells and loss of mineralized bone. It is estimated that about 10% of mineralized bone is loss per cycle. Furthermore, TSH is a negative regulator of bone remodeling, inhibiting the formation, the survival of osteoclasts and the differentiation of osteoblasts. Recent studies have shown that low TSH levels, *per se*, can lead to osteoporosis and fragility fractures. Hypercalcemia, hypercalciuria and a negative balance of calcium were also described. The weight loss and the gastrointestinal changes (decrease in intestinal calcium absorption and modified vitamin D metabolism) are also associated to the reduction of the body lean mass, thus inducing a higher risk of fragility fractures. In old and young Portuguese patients with endogenous hyperthyroidism, both men and women, significant decreases in the BMD in several skeletal regions and an increase in the prevalence of osteoporosis/low BMD were observed. Moreover, in young Portuguese men with hyperthyroidism, we found a trend for an increase in the prevalence of osteoporotic vertebral fractures detected by VFA. In a group of postmenopausal women with hyperthyroidism compared to a control group, we detected a significantly higher prevalence of reduced BMD at all skeletal sites and also of osteoporosis. Regarding subclinical hyperthyroidism in postmenopausal women, we found already significant correlations not only in bone turnover markers but also in some of the hormones implicated in bone metabolism.

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PP04. OSTEOPOROSIS IN MEN

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Introduction: The bone strength and fracture risk depend on several parameters: macrogeometry of cortical bone, BMD, trabecular bone microarchitecture, bone microdamage, bone mineralization, and bone metabolism. The trabecular bone score (TBS) of the Ukrainian men with osteoporotic vertebral fractures has not yet been studied.

Objectives: To evaluate TBS and BMD in men with osteoporotic vertebral fractures.

Methods: We've examined 243 men aged 30-89 years, divided according to the gerontologic classification: 30-44 yrs (n = 46), 45-59 yrs (n = 83), 60-74 yrs (n = 86), 75-89 yrs (n = 28). The basic group consists of 52 men with osteoporotic vertebral fractures in the anamnesis (mean age – 59.8 ± 13.7 yrs; mean height – 1.73 ± 6.98 m; mean weight – 79.0 ± 14.9 kg) and control group. of 191 men without fractures (mean age – 57.4 ± 13.7 yrs; mean height – 1.74 ± 6.89 m; mean weight – 76.5 ± 9.3 kg). BMD of PA lumbar spine and proximal femur were measured by the DXA method (Prodigy, GEHC Lunar, USA) and PA spine TBS were assessed by the TBS iNsight® software package installed on DXA machine (Med-Imaps, Pessac, France).

Results: We have observed a significantly lower TBS in the basic group (30-44 yrs – 1.083 ± 0.187, 45-59 yrs – 1.025 ± 0.248, 60-74 yrs – 1.084 ± 0.170, 75-89 yrs – 0.951 ± 0.170) as compared to the control group (30-44 yrs – 1.276 ± 0.121, 45-59 yrs – 1.226 ± 0.156, 60-74 yrs – 1.150 ± 0.175, 75-89 yrs – 1.183 ± 0.174); $F = 1.56$; $p < 0.001$. We also found the lower BMD of lumbar spine in the basic group of patients – 30-44 yrs – 0.981 ± 0.125 g/cm^2, 45-59 yrs – 1.028 ± 0.184 g/cm^2, 60-74 yrs – 1.014 ± 0.158 g/cm^2, 75-89 yrs – 0.970 ± 0.183 g/cm^2 ($F = 1.52$; $p < 0.001$) and of the proximal femur – 30-44 yrs – 0.854 ± 0.149 g/cm^2, 45-59 yrs – 0.873 ± 0.139 g/cm^2, 60-74 yrs – 0.823 ± 0.136 g/cm^2, 75-89 yrs – 0.716 ± 0.107 g/cm^2 ($F = 1.10$; $p < 0.001$) compared to the control group.

Conclusions: Subjects with vertebral fractures have TBS and BMD parameters significantly lower than the healthy men.

PP05. DIABETES MELLITUS AND BONE MASS

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The complications of diabetes mellitus (DM) and osteoporosis may cause severe morbidity and decreased longevity. Osteoporotic fractures are common in patients with low bone mineral density or with osteoporosis, but in DM the blindness and other eye problems, the hypertension, the cardiovascular disease, the amputations and other late complications such as chronic renal disease and neuropathy are also common. Bone is a multifunctional tissue with mechan-

ical, hematopoietic and metabolic tasks that result from the close interaction between the osteoblast and the osteoclast. Bone emerged lately as an endocrine organ controlling the metabolism of glucose. More recently, a special attention has been focused on the osteoblasts, osteoclasts and osteocytes functions in people with diabetes. Some complications of DM may lead to osteoporotic fractures. People affected with type 2 DM have increased fracture risk despite higher bone mineral density (BMD), as shown by trabecular bone score (TBS) and micro indentation of the tibia tests. Type 2 DM also results in an increased risk of fracture and delayed fracture healing. Since some years ago, some medications for type 2 diabetes were associated with an increase in bone fractures. Poor glycemic control in type 2 diabetes is associated with increased risk of fragility fractures. Increased longevity and a lifestyle characterized by low physical activity and high-energy food intake contribute to an increasing incidence of DM and osteoporosis. Both osteoporosis and DM have high social and financial costs. Hospital inpatient care, medication and supplies, retail prescriptions to treat complications, physician office visits and paramedical social assistance have high expenditures. The risk for precocious death among those with diabetes is about twice that of people with similar age but without diabetes. So, these important public health problems, DM and osteoporosis, are two of the leading causes of death. Thus, the bone tissue needs to be recognized as another important target among the late diabetic complications.

PP06. GENETIC RISK FACTORS FOR OSTEOPOROSIS IN UKRAINIAN POPULATION

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Introduction: Worldwide, osteoporosis and resulting fractures constitute a major public health burden with often devastating consequences, leading to increased levels of morbidity and mortality. Determination of molecular genetic causes of osteoporosis is a perspective approach. There are several methodologies to assess the contribution of a candidate gene in the pathogenesis of osteoporosis. One of them consists in determining the correlation between allelic polymorphism and candidate factors which cause the disease, which in comparison estimates allele frequencies of candidate genes in osteoporosis patients with individuals not having the disease and preserving normal bone mineral density.

Objectives: To determine the alleles frequency of genes. regulators of bone metabolism in patients with osteoporosis in Ukrainian population, and to assess the contribution of different polymorphisms in the risk of developing the disease.

Methods: DNA extraction was performed using the phenol-chloroform method from whole blood. Using PCR followed by restriction digestion and visualization of the reaction products in polyacrylamide gel have been studied 180 patients with osteoporosis and 160 healthy people of the same age.

Results: We have found association of polymorphism 60890 A/G of vitamin D receptor gene (OR = 3.2 (CI95% 2.2-4.6)) and -234 T/G polymorphism of collagen type 1 gene (OR = 2.8 (CI95% 2.1-4.1)) with the risk of osteoporosis developing. We have not found association of polymorphism -764 T/G of estrogen receptor gene (OR = 1,2 (CI95% 0.6-2.3)).

Conclusions: Knowing association between pathogenic alleles, candidate genes and osteoporosis in Ukrainian population will allow to use genetic testing to identify predisposition to the disease. The results of this study are important for a more rational organization of the prevention and treatment of the illness in the early stages of disease development.

PP07. OSTEOPOROSIS GENETIC STUDIES IN A SAMPLE OF A PORTUGUESE POPULATION

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Introduction: Several cardiovascular diseases (hypertension diabetes, atherosclerosis, heart failure, and chronic kidney diseases) have physiopathology pathways in common with osteoporosis.

Objectives: To evaluate the genetic variation in those pathways in association studies of osteoporosis.

Methods: The evaluation of osteoporosis and obesity was done in 650 subjects after measurement of anthropometric parameters like BMI (Kg/m^2) and BMD (g/cm^2) by DEXA. For the stratification of BMD, densitometry was evaluated with QDR Discovery W and the respective software 12.02 (Hologic Inc). Genotyping was performed for SNPs, Ins/Del and CNVs with PCR, PCR/RFLPs and PAGE technics. Statistics test were done with the used of SPSS programme. The analyses of continuous parameters and discrete data were employed with the appropriate tests.

Results: We studied variation in genes coding proteins belonging to acute phase subsystems (Renin–Angiotensin, Haptoglobin-HFE), hormonal systems (CYP1A1-COMT), neurotransmitters (5HTLPR, 5HTVNTR), transducing signals (ACP1 or LMWPTP) and metabolic pathways (MTHFR, GSTM and GSTT).

Conclusions: We observed some involvement of those genetic polymorphisms in the susceptibility to osteoporosis and its relationship with the metabolic syndrome, not only of individual genes but also for epistasis between them.

PP08. BONE METABOLISM AND PERIODONTAL DISEASES

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Introduction: Several studies discuss the relationship between systemic bone mineral density (BMD) and periodontal diseases. Osteoporosis or low systemic BMD should be considered as the risk factor for periodontal disease progression.

Objectives: The purpose of this study was to determine the impact of bone metabolism – bone formation, on the periodontal status in the patients.

Methods: The study included 72 patients (42 men and 30 women, mean age – 45.3 ± 6.2 years) with the healthy periodontal status (HPS) and 253 patients (145 men and 108 women, mean age – 45.6 ± 5.2 years) with generalized periodontitis (GP). Clinical conditions of periodontal tissue and radiographic determinations (panoramic X-Ray) were evaluated. Skeletal systemic BMD was measured by DXA. Metabolic processes of bone tissue were evaluated by bone turnover markers: bone tissue formation – osteocalcin (OC), bone-specific alkaline phosphatase (BAP) in serum.

Results: Comparative analysis of structural and functional state of bone tissue in patients showed a mineral density reduction in GP group compared to HPS, but these changes were not statistically significant. Disorders of bone tissue metabolism were determined in patients with GP. The OC level in the patients with GP (18.89 ± 0.87 ng/ml in men and 20.39 ± 1.14 ng/ml in women) was statistically significantly ($p < 0.01$) lower compared to the HPS group (24.14 ± 1.04 ng/ml in men and 27.56 ± 1.12 ng/ml in women). Decreased levels of bone formation of biochemical bone remodeling markers (BAP) were found in men (19.21 ± 1,76 U/l) and women (22.31 ± 1.65 U/l) GP compared to HPS (25. 21 ± 1.76 U/l in men and 29,17 ± 1,13 U/l in women, $p < 0.05$)