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, $p < 0.05$)

Conclusions: These results suggest that periodontitis associations with bone metabolic disturbances. In patients with periodontitis unbalanced bone remodeling was found: decreased bone formation. The results suggest the necessity to correct bone tissue metabolism in patients with generalized periodontitis by osteotropic medications.

PP09. MINERAL AND BONE DISTURBANCES IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Introduction: Patients with chronic kidney disease (CKD) frequently have disturbances in mineral metabolism, abnormalities in vitamin D. parathyroid hormone (PTH) homeostasis and bone disorders, known as CKD- MBD.

Objectives: To study the prevalence of bone and mineral metabolism disorders in patients with various stages of CKD.

Methods: We analyzed data on 220 adults with CKD aged 20-61 years. 78 (35.5%) patients with CKD II-IV stages and 142 (64.5%) patients on hemodialysis (CKD VD). BMD was measured using DEXA in L1-L4 segment and femoral neck. The laboratory investigations included PTH, vitamin D, calcium (Ca), phosphate (P) serum concentrations.

Results: The concentrations of Ca × P product and PTH were significantly higher in hemodialysis patients compared to CKD stages II-IV ones (Ca × P product 4.78 ± 0.11 vs 3.68 ± 0.18, $p < 0.01$, iPTH 601.28 ± 68.45 vs 289.10 ± 60.48 $p < 0.01$). Analyzing the compliance with KDIGO 2011 recommendations it was found that all four parameters met target levels of only in 14.1% patients with CKD II-IV stages and 3% with CKD VD stage. Vitamin D insufficiency was found in 44.9% of CKD II-IV patients and 51.4% those with CKD VD. BMD was decreased in 55.4% CKD II-IV stages patients and in 19.1% it was lower than -2.5 T SD. In CKD VD BMD was decreased in 51.1% and in 34.0% it was lower than -2.5 T SD. Negative association between decreased renal function and decreased BMD was established: GFR correlated s with spine-BMD ($r = -0.452$, $p < 0.05$).

Conclusions: In CKD patients dominant disorders of mineral metabolism are hyperphosphatemia, secondary hyperpathyroidism and 25 (OH) D_3 insufficiency. They occur in the early stages of CKD and progress with the decline of renal function, especially in hemodialysis and result in bone loss.

PP10. TRIKS AND PITFALLS IN DXA INTERPRETATION

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Introduction: Many factors can cause wrong results and incorrect interpretations in DXA. Assuming that a quality control program of the equipment is working, the causes rely mainly in the patient or in the interpretation.

Objectives: Identifying causes of reanalysis and repetition of DXA analysis in an outpatient practice.

Methods: We reviewed all the DXA examinations done in 6185 patients, made in 2 sites, with LUNAR DPX equipments, by a team of 12 technologists. Examinations were reported by two consultant radiologists (and also certified clinical densitometrists). Correlation with other imaging modalities (X-ray, CT, MR) was available on PACS in many cases.

Results: Being this poster a *pictorial assay*, the total number of cases or the full distribution are not relevant. We will present the more frequent identified errors and other rare ones: errors in data introduction, patient artefacts, anatomy variants, deficient position and coexisting diseases or therapeutic instrumentations (and not forgetting analysis mistakes...) The main objective in this presentation is to learn with our mistakes.

Conclusions: Particular care in checking that patient has really removed all artefacts and that hasn't done (or is going to make) another imaging technique. Don't rely in equipment anatomical detection. Any abnormal discrepancy should be checked and eventually additional X-ray image be done. We hope that this pictorial assay can help reduce the number of second examinations and difficult interpretations and alert to potential error situations.

PP11. TBS IN FRAGILITY FRACTURE RISK ASSESSMENT

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Objectives: The aim of the study was to evaluate the Bone Mineral Density (BMD), Trabecular Bone Score (TBS) and the 10-year probability of major osteoporotic fracture and hip fracture in healthy men of different ages.

Methods: We've examined 300 men aged 40-89 years. They were divided into groups depending on their age: 40-49 yrs (n = 52), 50-59 yrs (n = 86), 60-69 yrs (n = 89), 70-79 yrs (n = 59), 80-89 yrs (n = 14). The 10-year probability of hip fracture and the 10-year probability of major osteoporotic fracture risk were calculated by Austrian, Polish and Russian FRAX® models. BMD of whole body, PA lumbar spine and proximal femur were measured by DXA method (Prodigy, Lunar) and PA spine TBS were assessed by TBS iNsight® software package installed on the available DXA machine (Med-Imaps, Pessac, France).

Results: We have observed a significant increase of 10-year probability of major osteoporotic fracture in men aged 80-89 yrs ($p < 0.01$) by Russian FRAX® model, 60-89 yrs ($p < 0.01$) – Austrian FRAX® model, 70-89 yrs ($p < 0.01$) – Polish FRAX® model in comparison with men aged 40-49 yrs. 10-year probability of hip fracture was significantly increased in men aged 70-89 yrs in comparison with men aged 40-69 yrs ($p < 0.01$). It was determined the significant decreasing of TBS in men according to their age (40-49 yrs – 1.116 ± 0.02, 50-59 yrs – 1.111 ± 0.02; 60-69 yrs – 1.118 ± 0.02; 70-79 yrs. 1.062 ± 0.02, 80-89 yrs – 1.080 ± 0.05; $F = 2.42$, $p = 0.048$). TBS in men was significantly higher in subject with normal BMD (1.121 ± 0.01) compared with patient who osteoporosis – 1.066 ± 0.03 ($p = 0.04$). The significant correlation was observed between TBS and BMD L1-L4 in examined men ($r = 0.12$; $p = 0.03$). There wasn't any correlation between TBS and BMD of femoral neck.

Conclusions: TBS significantly decreased with ageing. Subjects with osteoporosis have significantly lower TBS compared with normal BMD examined. It was found a significant correlation between TBS and BMD L1-L4.

PP12. TRABECULAR BONE SCORE IN PORTUGUESE POPULATIONS

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The bone strength is mostly dependent on bone mineral density and microarchitecture (quality). The BMD by DXA scan is the gold