Figure 1. Lumbar Spine T-score and FRAX risk for OP

Figure 2. Femoral neck BMD and FRAX risk for Hip

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SAT0467 LOW SERUM OSTEOCALCIN LEVELS ARE ASSOCIATED WITH THE PRESENCE OF DIABETES MELLITUS IN GLUCOCORTICOID TREATED PATIENTS.

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Background: Increasing evidence indicates that osteocalcin (OC) is involved in the regulation of glucose homeostasis. Glucocorticoid (GC) treatment is associated with impaired osteoblast function and decreased OC levels and also with the development of GC-induced diabetes mellitus (GIDM). However, whether decreased OC levels in GC-treated subjects contribute to GIDM is not well known.

Objectives: To analyse whether OC levels in GC-treated patients are associated with the presence of GIDM.

Methods: 127 patients (aged 62±18years, 63% women) on GC treatment for autoimmune diseases (≥5mg/day, >3 months) were included. Clinical and anthropometric data were analysed, including the GC dose and treatment duration, presence of GIDM, fragility fractures, densitometric osteoporosis and bone formation (OC, bone alkaline phosphatase [BAP], PINP) and resorption markers (urinary NTX, serum CTX). The cut-offs of each bone marker for the presence of GIDM were estimated and optimized with the Youden index and included in the logistic regression analysis (adjusted for BMI, age and GC doses).

Results: 17.3% of patients presented GIDM. Diabetic subjects were older (70.5±12.2 vs. 59.6±18.4, p=0.001) and had a higher BMI than non-diabetics (30±5.2 vs. 26±4.2, p=0.002). No differences were observed in GC dose or duration or in the presence of vertebral fractures. Diabetics showed lower levels of OC (7.57±1.01 vs. 11.56±1; p<0.001), PINP (21.48±1.01 vs. 28.39±1; p=0.0048), NTX (24.91±1.01 vs. 31.7±1; p=0.036) and CTX (0.2±1.01 vs. 0.3±1; p=0.0016) with similar BAP values. The best discriminating cut-offs for GIDM presence were: <9.25ng/mL for OC, <24ng/mL for PINP, <27.5nMol/mM for NTX and <0.25ng/mL for CTX. On multivariate analysis OC (<9.25) was the only marker related to the presence of GIDM (OR 6.1; CI95% 1.87-19.89; p=0.001).

Conclusion: Decreased OC levels in GC-treated patients are associated with an increased risk of GIDM, a finding that was not observed with other bone turnover markers, further confirming the involvement of OC in the glucose homeostasis regulation in this entity.

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SAT0468

EFFECTS OF ANDROGEN DEPRIVATION THERAPY ON BONE QUALITY (TBS) IN PATIENTS WITH PROSTATE CANCER

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Background: Androgen deprivation therapy (ADT), by inducing severe hypogonadism, leads to a loss of bone mineral density (BMD) and an increased risk of fragility fractures after 6 months of treatment in men with prostate cancer¹. However, its effect on bone quality has not been described.

Objectives: To evaluate the changes on bone microarchitecture (bone quality) assessed by TBS (trabecular Bone Score) in male patients with prostate cancer after one year of treatment with ADT.

Methods: All patients diagnosed with prostate cancer candidates for long-term ADT admitted to Urology department of Hospital Universitari Parc Taulí (reference population of 450,000 inhabitants) between April 2017 and December 2019 were included. Patients who received chemotherapy, previous hormonal therapy or specific treatment for osteoporosis in the last year or those who had a very impaired functional capacity (Barthel index <30) were excluded. Demographic, clinical and analytical data (testosterone, calcium, phosphorous, alkaline phosphatase, 25-hydroxyvitamin D, PTH) were collected in all patients. A bone densitometry (GE-Lunar Prodigy) including the measurement of lumbar spine TBS (L1-L4) using Medimaps Software was performed at baseline and at 12 months of treatment with ADT.

Results: 78 patients were included. Mean age 77.9±8.3 years. The median Gleason score was 7.88±1.05. 3 patients had previous fragility fracture (one sacral fracture, one fibula and one multiple vertebral fracture). Baseline analytical values in patients were the following: testosterone 11.6±74.9 nmol/L; 25-hydroxyvitamin D 20.8±10.4 ng/ml; PTH 51.8±23.0 pg/ml; CTX 0.58±0.66. The daily calcium intake was 573±207 mg/day.

According to BMD, 17 patients (21.8%) had osteoporosis before starting ADT, with the following average T-score values: lumbar spine +0.15±1.85, femoral neck -1.75±1.00, and total hip -1.19±1.16. Mean baseline TBS value of the entire cohort was 1,279±0.122. 30.5% of the patients showed very degraded microarchitecture (TBS<1,230), 28.8% had partially degraded microarchitecture (TBS 1,230-1,310) and in 40.7% showed normal microarchitecture (TBS >1,310).

After one year of ADT treatment, TBS mildly worsened in this cohort, with a median value of 1,256±0.131 (p = NS). However up to 43% of patients reached highly degraded microarchitecture, 27% partially degraded and only 29.5% had a normal TBS (p = NS).

Conclusion: Most patients with prostate cancer have an altered bone quality before starting ADT. After 12 months of treatment, the percentage of patients with highly degraded bone microarchitecture increases, although not significantly. More studies are needed to confirm this trend and to evaluate if these patients present more long-term fractures.

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[1] Lee R, et al. Bone 2011; 48 (1): 88-95

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SAT0469

PROSPECTIVE STUDY ASSESSING BONE MINERAL DENSITY AND RISK FACTORS FOR OSTEOPOROSIS IN PATIENTS WITH ANDROGEN DEPRIVATION THERAPY. PRELIMINARY CROSS-SECTIONAL RESULTS

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Background: Few studies have analysed the incidence and risk factors for osteoporosis (OP) development in patients with prostate cancer (PC) and androgen deprivation therapy (ADT).

Objectives: To assess risk factors for OP, bone turnover markers (BTM) and bone mineral density (BMD) in a cohort of patients with ADT, as well as the duration of ADT and previous treatments received for PC.

Methods: Ongoing prospective study including patients with ADT for PC. Risk factors for OP, BTM (total ALP, bone ALP, CTx), spinal X-Ray and BMD (Lunar, DPX) were assessed yearly since inclusion in the study (April 2018). Patients with known OP or previous antiosteoporotic treatment were excluded. The study was approved by the ethics committee, and all patients gave their signed consent. Herein we present the preliminary cross-sectional study at inclusion.

Results: Of the 83 patients attended at the Rheumatology Department during the study period, 75 were included with a mean age 75±5 years and median ADT duration of 1 year. 18 were receiving concomitant radiotherapy and 7 docetaxel. When assessing risk factors for OP: 28% had previous fragility fractures and 24% had current alcohol intake. After X-Ray assessment, 14% had morphometric vertebral fractures. Mean 25OHD at inclusion was 19±9 ng/ml (73% had 25OHD <30 ng/ml) and mean testosterone was 82±162 ng/dL (75% had levels <50 ng/dL). All patients had increased values of CTx and 9% had increased bone ALP levels. BMD showed up to 28% with densitometric OP and osteopenia in 56%. Patients with OP were older (83±7 vs 74±8 years, p=0.021), had lower testosterone levels (16 vs 89 ng/dL, p=0.004), as expected lower BMD (at spine, proximal femur and even distal radius) and had more previous fragility fracture (75 vs 19%, p=0.022). But it should be noted that 16% had high bone mass (HBM) mostly affecting spine BMD (in 6 patients combined with femoral osteopenia). All patients with HBM had high bone metastatic disease, and no differences were observed between patients with/without HBM when comparing BTM or calcium-phosphate metabolism.

Conclusion: Low bone mass (including osteoporosis and osteopenia) is frequent in patients with ADT as well as previous fragility fractures. Up to 16% had high bone mass, being mostly in patients with high volume metastatic disease. Thus, all patients with ADT should undergo a bone health assessment and start antiosteoporotic treatment if required.

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SAT0470

FRAX AND DXA-BASED APPROACHES IN DIAGNOSTICS OF OSTEOPOROSIS RISK IN POSTMENOPAUSAL WOMEN WITH RHEUMATOID ARTHRITIS

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Background: Nowadays, the bone mineral density (BMD) measured by DXA and FRAX[®] are the most important methods for fracture risk assessment. Rheumatoid arthritis is a crucial disease for bone loss and osteoporosis development [1] which is included into FRAX algorithm. Ukrainian version of FRAX [2] is a new one and its value should also be assessed in patients with RA.

Objectives: Our study was aimed to assess the parameters of BMD and FRAX in postmenopausal women with RA.

Methods: We have examined 635 postmenopausal females aged 50-89 years old which were divided into 2 groups: 1st (control, n=313) – without any factors which have influence on bone metabolism, 2^d (n=322) – patients with RA. The 10-year probabilities of hip fracture and major osteoporotic fractures were

calculated without BMD parameter using the Ukrainian FRAX model [2]. The DXA was used to measure the lumbar spine, femoral neck and total body BMDs; the T-score was calculated (DISCOVERY Wi, Hologic, Inc., USA).

Results: FRAX indexes for major osteoporotic and hip fractures were significantly higher in patients with RA (Me [25-75Q]: 9.7 [6.8-15.0] and 3.1 [1.5-6.2] % compared to 6.0 [3.8-8.5] and 1.2 [0.6-2.4] % in females from the control group (p<0.001). 45.3 % of women with RA required antiosteoporotic treatment according to Ukrainian FRAX threshold ratio without measure of BMD compared to 12.1 % of subjects from control group. Only 3.4 % of patients with RA had FRAX indexes which were less than low threshold (requirement of additional DEXA measurement) compared to 31.3 % of females from control group.

BMD of femoral neck and distal radius were reliably lower in subjects with RA and consisted 0.65±0.13 and 0.69±0.12 g/cm in 1st and 2^d groups, accordingly (p<0.001) and 0.56±0.10 and 0.58±0.09 g/cm (p=0.02) without any significant differences at lumbar spine and total body BMDs. 16.6 % of subjects from the control group and 31.6 % of females with RA had osteoporosis according to DXA parameters (T-score ≤ -2.5 SD).

Conclusion: FRAX should be used more widely in clinical practice for detection of risk of osteoporotic fractures in subjects with RA.

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SAT0471

EFFECTS OF ONE YEAR TOFACITINIB THERAPY ON BONE DENSITY AND BIOMARKERS OF BONE TURNOVER IN RHEUMATOID ARTHRITIS

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Background: Oral JAK inhibitor, tofacitinib appeared as a new therapeutic option, beside biological therapies, which has already proven its safety and effectiveness in RA, but we lack of knowledge how it affects density of bone structures and bone turnover markers.

Objectives: The aim of this study was to assess the effects of one-year tofacitinib therapy on bone metabolism in patients with RA.

Methods: Altogether 30 RA patients with active disease were recruited and treated with tofacitinib in this 12-months follow-up study. Mean age of patients were 52.8±10.0 years, duration of rheumatoid arthritis were 7.7±5.0 years. Half of the patients haven't received biological treatment prior tofacitinib therapy, other half of the patients switched to tofacitinib therapy after completing washout. 15 patients received 2x5mg and 15 patients received 2x10mg tofacitinib daily for 12 months. On both arms 2-2 patients have discontinued treatment and excluded from the study. Assessments were performed at baseline, month 6 and 12. Levels of CRP and IgM rheumatoid factor (RF) antibodies were measured by quantitative nephelometry and levels of anti-CCP, sclerostin, osteocalcin (OC), P1NP were assessed by ELISA. Bone density was assessed by DXA (dual-energy X-ray absorptiometry, Lunar) and pQCT imaging techniques. Levels of DKK-1, OPG, RANKL were measured by multiplex microbead immunoassay (BioLegend LEGENDplex). In addition, disease activity (DAS28), age and disease duration were also measured. Correlations were determined by Spearman's analysis. Univariate and multiple regression analysis using the stepwise method was applied to investigate independent associations between DXA measurements (dependent variables) and laboratory parameters (independent variables).

Results: Tofacitinib significantly reduced DAS28 (p<0.001) and HAQ values (p=0.001), also level of CRP (p<0.001) and We (p=0.014). With respect to bone biomarkers we have experienced significant increase in levels of OC (p=0.013), OPG (p=0.006), sclerostin (p=0.026) and vitamin-D (p=0.017) at month 6, also in levels of OPG and vitamin-D (p=0.004, p=0.003) at month 12. We have found decrease in levels of CTX at month 6 (p=0.009) and 12 (p=0.003). When we examined the groups separately, we've found significant increase in levels of P1NP (p=0.027, p=0.005), OPG (p=0.005, p=0.002) and vitamin-D (p=0.001, p=0.004) at month 6 and 12, also in OC at month 6 (p=0.027) in Group A (2x5mg). In Group B (2x10mg) we've experienced a significant decrease in levels of phosphate and CTX at month 6 and 12 (p=0.012, p=0.021, and p=0.005, p=0.007).