practice, and it is very important to know risk factors that attenuate its effect in the treatment of osteoporosis.
Objectives: The aim of this study was to identify risk factors for inadequate response to the treatment of osteoporosis by DMAb.
Methods: Sixty-six patients treated with DMAb were observed retrospectively for one year. The mean age was 74 years, women were 91% and 36 patients with RA were included.We measured BMDs at lumbar and hip by dual-energy X-ray absorptiometry (Hologic Discovery) at baseline and one year later. We evaluated the effects of age, body mass index (BMI), use of glucocorticoide (GC), previous treatment for osteoporosis, BMD at baseline, bone metabolic markers (BAP; bone alkaline phosphatase, uNTx; urinary N-telopeptide), serum Ca and P levels and the previous vertebral fractures for inadequate response to DMAb.We defined the cases who could not gain the increase of BMD over 2% at the lumbar vertebrae and 4% at the hip as inadequate responders by taking the measurement error into account.
Results: Dose of PSL was significantly high in non-responder at non-RA trochanter and RA lumbar BMD (p=0.028, 0.006). BAP was higher in non-responder at RA lumbar BMD (p=0.007). Urinary NTX was significantly low in non-responder at non-RA lumbar and RA trochanter BMD (p=0.026, 0.048). Previous treatment for osteoporosis was significantly high in non-responder at non-RA lumbar, total hip and trochanter BMD (p=0.026, 0.022, 0.003).Multivariate logistic analysis including age, BMI, dose of PSL, BMD at baseline, BAP, uNTX, Ca and P level as confounders revealed that dose of PSL was the significant risk factors for no-response at lumbar BMD (OR0.634, 95% CI 0.433–0.93, p=0.02).
Conclusions: Patients receiving GC might not gain an adequate response to the treatment by DMAb for osteoporosis. Reducing dose of GC or altanative treatment regimen might be necessary.
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FRI0552 THE CORRELATION BETWEEN EROSION AND BONE MINERAL DENSITY IN PATIENTS WITH RHEUMATOID ARTHRITIS TREATED WITH DENOSUMAB AND BIOLOGIC DMARDS: A PROSPECTIVE COHORT STUDY

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Background: We have previously reported the efficacy of denosumab combined with biological DMARDs on radiographic progression in rheumatoid arthritis (RA).
Objectives: The aim of this work was to reveal the relationship between the changes in structural joint damage and in bone mineral density (BMD) in patients with RA who were started on denosumab in addition to biologic DMARDs.
Methods: We prospectively evaluated erosion (ERO) and joint space narrowing (JSN) scores by the van der Heijde-modified Sharp method and T-scores of the lumbar spine (LS) and total hip (TH) by dual energy X-ray absorptiometry scans at baseline and 12 months in the RA patients who were started on denosumab (60 mg every 6 months) for osteoporosis in addition to biologic DMARDs. We compared the 12-month change in ERO or JSN scores (∆ERO; ∆JSN) with the change in T-scores of the LS or TH (∆LS; ∆TH).
Results: Twenty-two patients (1 man and 21 women) at the mean±SD age of 74.4±8.1 at baseline were included in this study. The T-scores of the LS and TH at baseline were -1.38±1.57 and -2.53±0.85, respectively. ∆ERO, ∆JSN, ∆LS, and ∆TH were 0.16±0.49, 0.44±0.66, 0.30±0.39, and 0.20±0.42, respectively. BMD was significantly increased from baseline to 12 months. There was a significant inverse correlation between the ∆ERO and the ∆TH (ρ=–0.473, P=0.03), while there was no correlation between the ∆ERO and the ∆LS (P=0.98) nor correlation between the ∆JSN and the ∆LS (P=0.57) or ∆TH (P=0.25).
Conclusions: The change in erosive joint damage of hands and feet showed a significant relationship with the change in femoral BMD in patients with RA treated with denosumab and biologic DMARDs. BMD was ameliorated along with erosion by denosumab combined with biologic DMARDs, and may be an indicator for joint destruction.
References:
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FRI0553 THE EFFICACY OF 2-YEARS DENOSUMAB TREATMENT FOR GLUCOCORTICOID-INDUCED OSTEOPOROSIS (GIOP)

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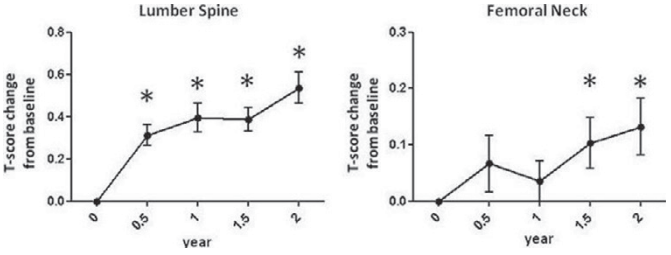
Background: Osteoporosis is one of the important adverse effects in the glucocorticoids treatment for the patients with rheumatoid arthritis (RA) and connective tissue diseases (CTDs). Although the usefulness of denosmab for primary osteoporosis has been well-established, the efficacy for GIOP remains unclear.
Objectives: This study aimed to clarify the therapeutic effects of denosumab for GIOP.
Methods: We evaluated bone mineral density (BMD) and serum markers of bone metabolism (BAP, NTx, TRACP-5b and P1NP) of patients who had been treated with over 5mg of predonisolone for RA and CTDs, and denosumab for GIOP, for two years in Kobe University Hospital. BMD and serum markers were evaluated every six months for 2 years from the baseline. The changes of those data from baseline were analyzed by Student's t test using GraphPad Prism 5 software and $p<0.05$ was considered statistically significant.
Results: Number of the patients were 53 (male: 4 cases, female: 49 cases), and their characteristics at the beginning of denosumab treatment were as below; age: 64.19±12.0 years old, dose of prednisolon: 10.59±9.97mg/day, BMD of lumber spine: 0.768±0.112g/cm^3, T-score of lumber spine: -2.28±1.01, BMD of femoral neck: 0.540±0.085g/cm^3, T-score of femoral neck: -2.28±0.76. After 2-years denosumab treatment, T-scores of lumber spine (0.54±0.39 gain) and femoral neck (0.13±0.26 gain) were significantly increased from baseline (Figure; mean ± SEM. *:$p<0.05$). In addition, the serum markers of bone metabolism, both absorption and formation, were significantly suppressed with denosumab.



Conclusions: Denosumab can suppress bone metabolic turnover, and increase lumber spine and femoral neck T-scores of GIOP patients.
Disclosure of Interest: None declared
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FRI0554 NEW BIOMARKER FOR DIAGNOSIS OF OSTEOPOROSIS IN RHEUMATOID ARTHRITIS PATIENTS

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Background: Bone mineral density and proteins/peptides determination in blood and urine as markers of bone resorption and formation are currently used to diagnose osteoporosis (OP) and metabolic bone diseases. However, these methods have some disadvantages for bone turnover evaluation. Recent evidence suggests that in RA changes in the secretion of hormones of white adipose tissue can be revealed [2]. One of them is Adiponectin possessing anti-inflammatory, anti-diabetic and anti-atherogenic properties [1]. Changes in Adiponectin levels may reflect influence of immune inflammation on bone turnover.
Objectives: To study the clinical and diagnostic value of serum Adiponectin determination in RA patients complicated by OP.
Methods: We examined 88 women with documented diagnosis of RA and mean disease duration of 6.56±0.88 years. We used EULAR/ARA 2010 criteria to diagnose the patients. Female patients with II degree of disease activity (DAS28), Steinbrocker stage II (erosive), rheumatoid factor- and anti-cyclic-citrullinated peptide antibody-positive were prevalent. We excluded patients who had surgery or developed an infection within the last 8 weeks, pregnant and breast-feeding women, those with severe heart, liver or kidney disease, immune deficiency, leukopenia or chronic infection.
A control group of 45 healthy females aged of 25 and 59 years were included in the study. There were no reported findings of joint pain and RA symptoms in the group. The groups were adjusted for age ($p>0.05$) and showed no statistically significant differences.
We measured serum Adiponectin levels (μg/ml) using Human Adiponectin ELISA commercial test systems. We plotted a curve using computer software. We diagnosed OP using dual-energy X-ray absorptiometry with LUNAR DPX PRO (GE, USA).
Results: Serum adiponectin levels in the control group were 12.5±0.9 μg/ml (M±m). Adiponectin levels in healthy subjects measured as M ± 2d, ranged between 0.44 and 24.56 μg/ml. Patients with OP and RA had significantly higher levels of serum Adiponectin ($p<0.001$). Mean serum Adiponectin levels in RA patients who had normal bone density and had no OP were 35.21±0.6 μg/ml. Mean serum Adiponectin levels in RA/OP patients with low bone mineral density were 52.42±0.69 μ g/ml. Adiponectin levels of 44 μg/ml and higher were