CAN WE AVOID THE LOSS OF BONE MINERAL IMPACT OF GLUCOCORTICOID TAPERING ON

Table 1. Sample’s characteristics

<table>
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<th>Total (N)</th>
<th>437</th>
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Age at admission, y (median, min-max): 83 (65-101)
Sex, female (%): 368, 79.7
Degree of dependence (N): 288
Total dependent (%): 20, 7.0
Partially dependent (%): 76, 17.4
Autonomous (%): 192, 66.7
Type of fracture (N): 427
Subtrochanteric (%): 36, 8.2
Femoral neck (%): 176, 40.3
Transtrochanteric (%): 225, 51.5
Type of intervention (N): 437
Girdlestone (%): 1, 0.2
No surgery (%): 6, 1.4
Total hip replacement (%): 68, 15.6
Partial hip replacement (%): 68, 15.6
Osteosynthesis (%): 292, 66.8
Destination at discharge (N): 437
Home (%): 254, 58.1
NH (%): 62, 14.2
RU (%): 121, 27.7
Vertebral fracture (N): 229
0 (%): 96, 41.9
1 fracture (%): 54, 23.5
≥ 2 fractures (%): 79, 34.5
Femur neck BMD, g/cm² (mean, SD, N): 0.7 ± 0.12, 205
Femur neck T score (mean, SD, N): -2.8 ± 1.04, 205
≥ 2.5 (%): 144, 70.0
≥ 4D, ng/ml (mean, SD, N): 23.5 ± 11.69, 210

Conclusion: Previous studies showed lower survival in patients discharged for conventional home care (1,2). In our cohort, despite more than half the sample was discharged home, no difference was found in mortality or re-fracture. Further studies will assist to clarify this matter.

REFERENCE:

Disclosure of Interests: None declared

Results: 71 post-menopausal women stopped denosumab after 7.7±2.2 injections: age 63.8±8.1 years, BMI 23.8±4.5, 0.96 prevalent fractures/patient, 8.45% previously exposed to corticoids, 22.54% to anti- aromatases. 17.25 months after last denosumab injection 30 patients were classified as Loosers and 41 as Stable. At denosumab introduction Loosers were younger (61.4±7.3 vs 65.5±8.2 years, p=0.034) with higher CTX level (64.7 vs. 47.4 ng/ml, p=0.005). The rate of BP given less than 2 years before denosumab was not different, but none of the Loosers had received zoledronate vs. 12% of the Stable (p=0.047). Other pre-denosumab characteristics were not different. Number of denosumab injections, BTMs and BMD values were comparable in both groups during denosumab treatment. First BTMs values measured 7.5 months (median) after last denosumab injection and before phosphonates were not different (sCTX: Loosers, 592 ng/ml; Stable, 379 ng/ml, p=0.06). At DD 59% received zoledronate, 24% iodendronate, 3% others, and 14% nothing (p=0.39 between groups). BTMs 12.8 months post-BP were higher in Loosers as compared to Stable (sCTX 537 vs. 356 ng/ml, p=0.009). Incidence of new fractures was low (0.18/patient) without between group’s difference.

Conclusion: In our sub-cohort, being younger, having high BMTs and not having received zoledronate before denosumab introduction increases the risk of a BMD loss, even if a bisphosphate is prescribed at DD. Our results support the use of denosumab after a bisphosphate to restrain the BMD loss at its discontinuation.

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Amen Navigant, Giovanni Liebic: None declared, Delphine Stoll: None declared, Elena Gonzalez-Rodriguez: None declared, Didier Hans Shareholder of: co owner of TBS, medimaps group, Speakers bureau: Amgen Lilly
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Speakers bureau: Amgen Lilly


IMPACT OF GLUCOCORTICOID TAPERING ON MARKERS OF BONE METABOLISM IN PATIENTS WITH RHEUMATOID ARTHRITIS WHO ACHIEVED LOW DISEASE ACTIVITY OR REMISSION ON TOCILIZUMAB: EXPLORATORY ANALYSIS FROM A RANDOMIZED CONTROLLED TRIAL

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Background: Glucocorticoids (GCs) directly impact bone metabolism via increased bone resorption and inhibited bone formation; hence, systemic fracture risk increases with daily and cumulative GC doses.2 However, many patients with established rheumatoid arthritis (RA) receive long-term treatment with GCs to suppress inflammation, which confers some benefit in delaying bone erosion.3 Objectives: This exploratory analysis of the SEMIRA (Steroid Elimination In RA) study4 compared changes in markers of bone and cartilage metabolism in RA patients with low disease activity (LDA) or remission on tocilizumab (TCZ) + GC vs conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) who received either slow GC tapering (GCtaper) or continuation (5 mg; GC5mg). Methods: Patients had at least stable LDA (DAS28-ESR ≤3.2) for ≥4 weeks, were receiving a stable prednisone regimen (5 mg/day) + TCZ + csDMARDs for ≥4 weeks, and received TCZ + GCs (prednisone equivalent 5-15 mg/day) for ≥6 months before randomization. Patients were randomly assigned to GC5mg (n=128) or GCtaper (n=131; starting at 4 mg/day with 1-mg reduction every 4 weeks to 0 mg/day at weeks 16-24) for 24 weeks, during which TCZ + csDMARDs remained stable. Change between baseline (BL) and week 24 in serum levels of markers of bone and cartilage metabolism—including N-terminal propeptide of type 1 collagen (P1NP), C-terminal telopeptide of type 1 collagen (CTX1), alkaline phosphatase (ALP), matrix metalloproteinase 3 (MMP3), calcium, phosphorus, and albumin—were exploratory outcomes. Within-group and between-group changes from baseline were evaluated by Wilcoxon paired rank test and Wilcoxon rank sum test, respectively.

Results: Figure 1 shows changes from BL to week 24 in bone and cartilage biomarkers. MMP3 (marker of cartilage degradation) was reduced at week 24 in the GCtaper group compared with GC5mg. Change from BL at week 24 in P1NP (bone formation) and CTX1 (bone resorption) favored the GCtaper arm, with P1NP increasing relatively more than CTX1 (44% and 24%, respectively, compared with GC5mg), indicating net anabolism. The increase in ALP from BL to week 24 was lower in the GCtaper arm than for the GC5mg arm, suggesting neomineralization. There was no difference in the change from BL to week 24 in calcium, phosphorus, or albumin (not shown).
Conclusion: The findings of this biochemical marker analysis suggest that withdrawal from GC after achievement of LDA or remission with TCZ results in decreased bone remodeling, with a trend toward an anabolic window and reduced reversible risk of systemic harm to noninflamed bone versus benefits for inflamed joints in the context of LDA or remission.

REFERENCES:


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When rheumatoid arthritis (RA) does not walk alone: new data on comorbidities in RA

OP0088 COMORBIDITIES AS RISK FACTORS FOR RHEUMATOID ARTHRITIS (RA) AND ACCURACY AFTER RA DIAGNOSIS

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Background: Understanding the timeline of comorbidity development in patients with RA may inform disease pathogenesis and help identify targets for improving outcomes (1).

Objectives: We first aimed to compare the prevalence of a comprehensive list of comorbidities in RA cases versus controls. Second, we aimed to investigate the time association of comorbidity development relative to RA onset to identify which comorbidities might predispose to developing RA and comorbidities that might result from RA.

Methods: We performed a case-control study using a biobank at a single center, identifying 821 cases of RA (143 incident) using a rules-based algorithm combining two diagnosis codes with use of a DMARD (PPV = 95%). We matched each case to three controls based on age, sex, and location of residence at the time of the biobank survey. Participants self-reported the presence or absence and age of onset for 77 comorbidities on the survey.

Results: The prevalence of comorbidities among RA cases was higher than controls, with the most common being cardiovascular disease, obesity, depression, and anxiety. The time of comorbidity development varied by comorbidity, with some developing before RA onset and others developing after diagnosis. The accuracy of diagnosis varied by comorbidity, with some diagnoses being more accurate than others.

Conclusion: There appears to be a trend in the proportion of comorbidities developing before RA onset, with some developing within the first year after diagnosis and others developing more gradually. This information may help guide targeted interventions to prevent or mitigate the development of comorbidities in RA patients.