medication were: performing DXA scan (aOR = 9.9, 95% CI [7.7, 12.6]), fragility fracture (aOR = 2.8, 95% CI [1.8, 4.5]), and osteoporosis or osteopenia diagnosis (aOR = 2.5, 95% CI [2.0, 3.1]).

Conclusion: We describe temporal trends in bisphosphonate discontinuations and factors associated with discontinuation of alendronate and restart of osteoporosis therapy. From 2010 to 2015, approximately one-third of women with long-term bisphosphonate treatment discontinued treatment for ≥ 12 months. We observed an increasing trend in discontinuation from 2010 to 2012, which remained steady through 2015. Factors associated with discontinuation of alendronate were associated with worsening of overall health status, while factors traditionally associated with worsening bone health were associated with restart of osteoporosis medication.

Figure 1. Temporal trends of Bisphosphonate Discontinuation in U.S. Medicare women beneficiaries by calendar year

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OP0083

PREVALENCE OF VERTEBRAL FRACTURES IN POSTMENOPAUSAL WOMEN WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a risk factor for the development of fragility fractures, but there is little quality data on its prevalence. Conversely, osteoporosis is one of the most frequent comorbidities of RA.

Objectives: To determine the prevalence of vertebral fractures in postmenopausal women with RA and to analyse their characteristics and associated risk factors.

Methods: We included 346 postmenopausal women diagnosed with RA according to the ACR/EULAR 2010 criteria in 19 Spanish Rheumatology Departments, randomly selected from the registry of RA patients in each center, recruited during 2018. Lateral radiographs of the dorsal and lumbar spine were obtained from all patients, to evaluate morphometric vertebral fractures. Expert rheumatologists identified vertebral fractures and classified them into mild (grade 1: reduction of height of 20-25%), moderate (grade 2: reduction of 26-40%) and severe (grade 3: reduction > 40%), according to the Genant grading scale. The spinal deformity index (SDI) was calculated by assigning numbers 1, 2 and 3 to each fractured

vertebra and adding the total score of each patient. The study variables were: a) age, body mass index (BMI), b) factors related to RA: time of evolution, FR, ACPA, and c) fracture risk factors: prior fragility fracture, parental hip fracture, glucocorticoids, smoking, alcohol intake ≥ 3 units daily, secondary osteoporosis and time since menopause.

Results: The mean age was 66.8 (SD: 10.1) years and the median evolution of the disease, 8.00 [RIQ: 3.00-15.5] years. 77.2% (n: 267) and 75.7% (n: 252) had FR and ACPA +, respectively. The mean duration of the postmenopausal period was 15.0 (SD: 9.6) years. 23.4% (n: 79) of patients had at least one vertebral fracture; 10.7% (n: 36) had a single fracture and 12.7% (n: 43), multiple fractures. The most fractured vertebrae were D12, L1 and L2 (fractured in > 5% of patients). The median SDI was 3 [RIQ: 2-5]. The vertebrae with the highest mean IDE were D8, D10, D11 and L1 (all mean IDE ≥ 2). An association was found between the presence of vertebral fractures and age, height, postmenopausal period, time of disease progression, glucocorticoid treatment and parental hip. No linear association was found between SDI and age, time of evolution of the disease, BMI and time since menopause.

Conclusion: One out of every 4 postmenopausal women with RA has at least one vertebral fracture. Vertebrae of the dorso-lumbar hinge are the most frequently involved and the magnitude of the spinal deformity is relevant. Vertebral fractures are related to the time of evolution of RA and to the risk factors for fracture.

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OP0084

FRAGILITY HIP FRACTURES – DOES DESTINATION AFTER HOSPITAL DISCHARGE HAVE AN IMPACT ON PATIENT'S OUTCOME?

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Background: In Osteoporosis, hip fractures account for significant disease burden, with increased morbi-mortality. Because of this, patients are referred to rehabilitation and specific health care units, in order to regain autonomy and health. However, there is few evidence about patients' outcome, considering destination after inpatient discharge.

Objectives: To evaluate if destination after hospital discharge for a osteoporotic hip fracture has impact in the outcome (mortality and new fracture).

Methods: Patients referred to a Fracture Liaison Service from March 2015 until March 2017 with a fragility hip fracture were considered for this study. Clinical/demographic variables were retrospectively collected until January 2018, including destination at discharge (home, nursing home (NH), inpatient rehabilitation unit (RU)) and outcome (death, new fracture, lost to follow-up). Kaplan Meyer curves with log-rank test were used for statistical analysis (SPSS statistics 23); $p < 0.05$ was considered statistically significant.

Results: From a total of 522 patients, 437 patients were discharged with a known destination: 58.1% to home (n=254), 27.7% to a RU and the remaining to a NH. Table 1 summarizes clinical and demographic characteristics of the sample. Seventy-four patients died (median time = 264 days, range 22-1049) and 43 suffered a new fracture (median time 301, 15-946); 129 patients maintained follow-up. Mortality rates within each group of discharged patients were: 15.4% in the group destined home, 22.6% in the group forwarded to a NH and 17.4% in those referred to a RU. No statistically significant difference was found in cumulative survivals between groups (figure 1A). The event refracture was observed in 10.2% of those discharged home, 6.5% in the group forwarded to a NH and 10.7% of those referred to a RU. Again, no statistically significant difference was observed (figure 1B).

Table 1. sample's characteristics

Total (N)	437
Age at admission, y (median, min-max)	83 (65-101)
Sex, female (n,%)	368, 79.7
Degree of dependence (N)	288
Totally dependent (n,%)	20, 7.0
Partially dependent (n,%)	76, 26.4
Autonomous (n,%)	192, 66.7
Type of fracture (N)	437
Subtrochanteric (n,%)	36, 8.2
Femoral neck (n,%)	176, 40.3
Transtrochanteric (n,%)	225, 51.5
Type of intervention (N)	437
Girdlestone (n,%)	1, 0.2
No surgery (n,%)	6, 1.4
Total hip replacement (n,%)	68, 15.6
Partial hip replacement (n,%)	68, 15.6
Osteosynthesis (n,%)	292, 66.8
Destination at discharge (N)	437
Home (n,%)	254, 58.1
NH (n,%)	62, 14.2
RU (n,%)	121, 27.7
Vertebral fracture (N)	229
0 (n,%)	96, 41.9
1 fracture (n,%)	54, 23.5
≥ 2 fractures (n,%)	79, 34.5
Femur neck BMD, g/cm ² (mean, SD, N)	0.7, ±0.12, 205
Femur neck T score (mean, SD, N)	-2.8, ±1.04, 205
≤2.5 (n,%)	144, 70.0%
vitD, ng/ml (mean, SD, N)	23.5, ±11.69, 210

Figure 1. Panel A - outcome mortality; Panel B - outcome refracture.

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.

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OP0085

CAN WE AVOID THE LOSS OF BONE MINERAL DENSITY ONE YEAR AFTER DENOSUMAB DISCONTINUATION? THE REOLAUS BONE PROJECT

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Background: Denosumab discontinuation (DD) induces bone turnover markers (BTMs) increase, bone mineral density (BMD) decrease, and increased risk of spontaneous vertebral fractures. Prescribing a bisphosphonate after DD could avoid this rebound effect.

Objectives: The objective of the ReoLaus (Rebound Effect Observatory in Lausanne) Bone Project is to follow bone parameters after DD. We report the determinants associated to BMD loss 1 year after DD.

Methods: 170 patients are followed in ReoLaus. Patients with a BMD follow-up ≥ 1 year after DD with a standardized management were included. We defined as significant a lumbar spine BMD loss (Loosers group) over 3.96% at one year after DD as compared with values at the end of the denosumab treatment 18 months after last injection.

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 sCTX level (644.7 vs. 474.1 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 bisphosphonates were not different (sCTX: Loosers, 592 ng/ml; Stable, 379 ng/ml, p=0.06). At DD 59% received zoledronate, 24% alendronate, 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. 336 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 bisphosphonate is prescribed at DD. Our results support the use of denosumab after a bisphosphonate to restrain the BMD loss at its discontinuation.

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OP0086

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 formation¹; 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 ± 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 signed rank 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 greater for 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).