pulmonary disease and neoplasms vs biologic-naïve pts. Most biologic-failure pts (96.7%) had previously received ≥ 1 TNF inhibitor (TNFi): 48.7% had received 1 and 48.0% ≥ 2 TNFi; 56.6% had received ≥ 2 bDMARDs. The overall 2-year retention rate was 47.9% and was higher for biologic-naïve vs biologic-failure pts (54.5 vs 45.2%; $p<0.001$); the most common reasons for ABA discontinuation were inefficacy (61.4 vs 67.7%) and safety (21.3 vs 21.2%). In pts who discontinued ABA, 83.0% started a bDMARD ≤ 6 months after discontinuation (Table), most commonly ABA IV. Mean (SD) days from stopping ABA to starting a bDMARD was similar for biologic-naïve (93.4 [51.3]) and biologic-failure pts (93.6 [48.0]). Among pts who restarted ABA, 62 (80.5%) biologic-naïve and 158 (85.0%) biologic-failure pts were considered to have discontinued as the time from last dose was >84 (IV) or >28 (SC) days, and thus were no longer temporary discontinuations, as predefined in the protocol. Three pts discontinued for bad compliance, 3 for lack of efficacy, 3 for remission/major improvement, 12 for safety and 15 for surgery. A good/moderate EULAR response was achieved by 76.7% of pts at the last follow-up before ABA discontinuation and 58.3% at ABA restart; mean (SD) DAS28 (CRP) was 3.2 (1.1) and 3.8 (1.4), respectively.

	bDMARD ≤ 6 months after ABA discontinuation		bDMARD prior to initial ABA treatment in pts who restarted ABA	
	Biologic naïve n=186	Biologic failure n=526	Biologic failure n=186	
None	35 (18.8)	86 (16.3)		
Abatacept	77 (41.4)	186 (35.4)	–	
IV	71 (38.2)	170 (32.3)		
SC	6 (3.2)	16 (3.0)		
TNFi	41 (22.0)	74 (14.1)	181 (97.3)	
Adalimumab	10 (5.4)	12 (2.3)	108 (58.1)	
Etanercept	13 (7.0)	21 (4.0)	125 (67.2)	
Infliximab	7 (3.8)	13 (2.5)	55 (29.6)	
Certolizumab	7 (3.8)	17 (3.2)	5 (2.7)	
Golimumab	4 (2.2)	11 (2.1)	2 (1.1)	
Other bDMARD	33 (17.7)	180 (34.2)	51 (27.4)	
Anakinra	1 (0.5)	4 (0.8)	5 (2.7)	
Rituximab	6 (3.2)	58 (11.0)	30 (16.1)	
Tocilizumab	26 (14.0)	118 (22.4)	21 (11.3)	
	Data are n (%)			

Conclusions: Prior to abatacept treatment, over half of biologic-failure pts had received ≥ 2 bDMARDs and most had received a TNFi. After initial discontinuation (protocol defined), over one-third of pts restarted abatacept.

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AB0268 **ACPA SEROPOSITIVITY AND PERIPHERAL NATURAL KILLER CELLS AS PREDICTIVE MARKERS OF CLINICAL RESPONSE TO RITUXIMAB IN RHEUMATOID ARTHRITIS PATIENTS**

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Background: The efficacy of B cell-depletion therapy confirms the importance of B lymphocytes in rheumatoid arthritis's (RA) pathogenesis. Rheumatoid factor (RF) and anti-citrullinated peptide antibodies (ACPA) are prognostic factors for a more severe disease. Others immune elements, namely natural killer (NK) cells, seems to influence RA clinical response to rituximab (RTX), but data are lacking.

Objectives: To analyze the influence of baseline status/levels of RF, ACPA and serum immunoglobulin G (IgG) level in RTX treatment. To study the effect of RTX on NK and CD19+ cells in RA patients and their association with clinical response at 6, 12 and 18 months (M).

Methods: An observational retrospective study was conducted, including all the consecutive patients with diagnosis of RA under rituximab, followed at our Rheumatology department. Demographic and clinical data were obtained by consulting the national database (Reuma.pt) and the analysis was limited until december 2016. RF, ACPA and IgG titres were evaluated at baseline. NK (CD56+CD16+) and B lymphocytes (CD19+) absolute counts were assessed by flow cytometry prior to the first RTX cycle and 6 M after. Clinical responses were assessed by DAS28 and EULAR criteria at 6, 12 and 18 M. Correlations were

studied using Spearman coefficient analysis (SPSS 20.0). Significance level was set as 0.05.

Results: We included 63 RA patients (81% of women), with a mean (SD) age of 61 (10) years and a mean disease duration of 19 (10) years, 86% RF-positive and 87% ACPA-positive. Bone erosions were present in 86% of the patients. At baseline, the mean DAS28 was 5.79 (1.55). Combination therapy with methotrexate or with others cDMARDs was used in 48% and 30% of the patients, respectively; RTX monotherapy in 22% of our sample. Thirty three patients were previously exposed to other biologics. The magnitude of response was greater in ACPA-positive vs ACPA-negative patients in terms of DAS28 variation at 6, 12 and 18M (medians of 1.09 vs -0.08; 2.03 vs 0.35 and 2.10 vs 0.19; $p=0.029$, $p=0.039$ and $p=0.004$, respectively), without significant differences between groups in terms of initial DAS28 (5.91 (1.60) vs 5.00 (0.90), $p=0.051$). The presence of ACPA was also significantly associated with EULAR response at 6, 12 and 18 M (64%, 75% and 85% in ACPA-positive patients vs 25%, 16% and 25% in ACPA-negative patients; $p=0.034$, $p=0.010$ and $p=0.001$, respectively). Outcomes did not differ according FR status. There were no associations between the values of FR, ACPA and IgG at baseline with the clinical response (DAS28 variation). CD19+ cells depletion occurred in all patients (mean of 146.4/mm³ at baseline vs 10.6/mm³ at 6M). An increase of peripheral NK cells was seen at 6M (mean 231mm³ at baseline vs 289/mm³ at 6 M). We only have found a positive correlation between NK cells number at baseline and DAS28 variation at 6 M ($r=0.35$, $p=0.023$). There were no associations between, neither NK cells, nor CD19 cells variations at 6M with clinical response to RTX.

Conclusions: Our data suggest that ACPA seropositivity is associated with a better clinical response to RTX in RA patients. NK cells at baseline may be useful to identify early responders to RTX.

Disclosure of Interest: None declared

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AB0269 **RAPID RADIOGRAPHIC PROGRESSION PROGNOSTIC FACTORS IN A LATIN AMERICAN COHORT OF RHEUMATOID ARTHRITIS**

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Background: Rapid Radiographic Progression (RRP) in Rheumatoid Arthritis (RA) predicts long-term disability (1), high related economic costs (2) and loss of working time (3). Detection of RRP predictors as a necessary tool for aggressive therapeutic interventions has been proved, but in Latin-American cohorts, no available data had been reported.

Objectives: To determine independent risk factors associated with RRP in a cohort of RA patients

Methods: A prospective analysis of RA Almenara cohort (January 2015-April 2016), 500 patients followed up with annual evaluations (background clinical/epidemiology, clinimetric, laboratory, health questionnaires and X-rays) who meet ACR 87/ACR-EULAR 2010 criteria's, older than 16 years at diagnosis,sign informed consent. Patients with overlap (except Sjögren), with active infections and/or pregnancy are excluded. Patients with at least two radiographs (baseline/final) were included. The X-rays were taken in a standardized protocol (each hand and foot). Joint damage was measured by Sharp-VDH (erosions, decreased joint space, and total scores). Radiographic Progression (RP) as a annual difference in Sharp-VDH score and RRP (>5 units in RP) were determined. A blinded rheumatologist for the RA condition read all films. Associated factors were analyzed (gender, socioeconomic level, smoking, age at diagnosis, time disease, use of biological and non-biological DMARDs, DAS 28, current steroids, HAQ/functional capacity, RF and basal SharpVDH score). Diagnosis delay and CRP were included to analyze risk factors in the RRP group. Statistical analyzesTweedie regression models with logarithm link. SPSS v. 21.0 was used

Results: 153 patients, 90.8% women, middle low (37.9%) and middle (35.3%) the most prevalent socioeconomic status. Age at diagnosis was 46.06 (12.73) years, time disease 14.25 (10.26) years. DAS28 average: 4.51 (1.33). Basal Sharp VDH:104.53 (90.09), CRP: 9.77 (10.35) UI/L, RF: 352.49 (538.08) UI/L, ACPA: 563.64 (782.2)UI/dL. PR annual rate was 7.64 (2.4 years of follow up),94.8% increased damage and 74 patients (48.36%) had PRR. Most subjects (94.1%) were using DMARDs but only 15 (9.8%) biologicals. The mean dose of prednisone was 4.88 (3.33) mg/dl. In the univariate analysis, only DMARDs was associated with RP (B=50.8; CI=40.13–51.48, $p<0.001$), remaining this association in the multivariate analysis (B=50.56;CI=49.80–51.33, $p<0.001$). In the multivariate analysis of RRP group, no variables was associated with risk and subjects with steroids use had less radiographic impairment (B=0.859; CI 0.759–0, 893, $p=0.017$).

Conclusions: In this prospective study of an established RA cohort, a large proportion of patients had RRP and there was few percentage of biologic use. In the RRP subset only steroids use was a protective factor.

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