

**AB0207 CHANGE IN ANTI-CITRULLINATED PROTEIN AUTOANTIBODY LEVELS IN CLINICAL PRACTICE ARE ASSOCIATED WITH RESOURCE USE AND DISEASE ACTIVITY MEASURES**

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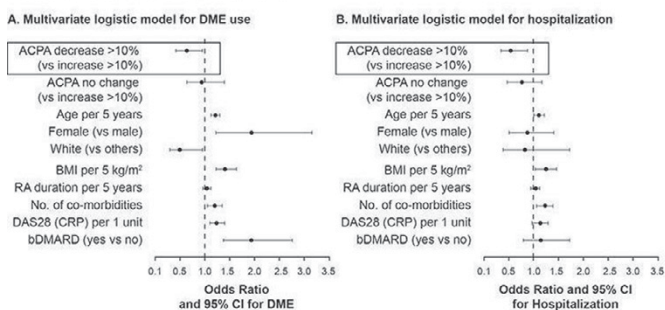
**Background:** High anti-citrullinated protein antibody (ACPA) concentration, beyond ACPA positivity, is indicative of more aggressive radiographic progression in patients (pts) with RA.<sup>1</sup> However, there is limited information on changes in ACPA levels in clinical practice settings, and the association of changes in ACPA with measures of resource use and/or disease activity.

**Objectives:** To evaluate the association between change in ACPA levels with hospitalizations/durable medical equipment (DME) use and change in disease activity.

**Methods:** Pts enrolled in a tertiary care centre RA registry, established in 2003, were analysed. The registry mostly comprises pts with established RA who were evaluated semi-annually for multiple clinical patient-reported outcomes as well as resource utilization parameters, and annually for disease activity measures such as DAS28 (CRP), SDAI and CDAI. The current analysis is based on pts enrolled in the registry with ACPA values at the time of baseline (BL) and follow-up visits. BL and follow-up ACPA levels were based on well-documented and validated ELISAs from Euro-Diagnostica (distributed by IBL-America, Minneapolis, MN, USA). Annual mean ACPA change from BL over the first year of enrolment in the registry was calculated. Changes ( $[(\text{follow-up} - \text{BL})/\text{BL} \times 100]$ ) in ACPA levels were categorized as decrease ( $<-10\%$ ), no change ( $-10\%$  to  $+10\%$ ) or increase ( $>+10\%$ ). Use of DME (canes, wheelchairs, walkers and commodes) as well as hospitalizations during 12-month follow-up and annual change in disease activity (DAS28 [CRP], SDAI, CDAI, swollen painful joint counts and pain) were assessed. Multivariate logistic regression analyses for binary outcome variables (DME and hospitalizations) and linear regression for change in disease activity measures were conducted, controlling for BL covariates.

**Results:** A total of 840 (65%) pts in the registry had BL and follow-up ACPA values and were included in the analysis. Overall, 34.6% (n=291) of pts had a decrease, 31.7% (n=266) had no change and 33.7% (n=283) had an increase in ACPA levels. There were no significant differences in BL characteristics between the three groups except for disease duration. Pts with RA with an increase in ACPA levels had significantly longer disease duration at BL. In univariate analyses, DME use was 23.4%, 30.1% and 28.6%, and hospitalization rate was 13.4%, 16.5% and 20.1% in pts with a decrease, no change or an increase in ACPA levels, respectively. Unadjusted mean (SD) change from BL in DAS28 (CRP), SDAI and CDAI in pts with reductions in ACPA levels was  $-0.7$  (1.4),  $-6.1$  (15.7) and  $-5.9$  (15.1), and  $-0.5$  (1.4),  $-5.0$  (15.7) and  $-4.7$  (14.6) in pts with an increase in ACPA levels. After controlling for BL covariates, the odds ratio (OR) for DME in patients who had a decrease in ACPA levels (vs increase) was 0.62 (95% CI 0.40, 0.94;  $p=0.026$ ), and the OR for hospitalization was 0.54 (0.33, 0.86;  $p=0.010$ ). Similarly, reductions in ACPA levels were associated with greater reductions in disease activity measures (Fig).

Figure. Association of Reductions in ACPA Levels With Resource Use (Panel A and B) and Disease Activity (Panel C) in Clinical Practice



\*Other variables in the models included age, sex, race, BMI, RA duration, co-morbidities, biologic use and baseline disease activity. ACPA=anti-citrullinated protein antibody; bDMARD=biologic DMARD; DME=hospitalizations/durable medical equipment; VAS=visual analogue scale

**Conclusions:** Reductions in ACPA levels were associated with reductions in DME use and hospitalizations as well as reductions in disease activity measures.

**References:**

[1] Ronnellid J, et al. Ann Rheum Dis 2005;64:1744-9.

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Employee of: Bristol-Myers Squibb, C. Iannaccone: None declared, M. Frits: None declared, N. Shadick Grant/research support from: Bristol-Myers Squibb, UCB, Mallinckrodt, Amgen, Crescendo Biosciences, Consultant for: Bristol-Myers Squibb, M. Weinblatt Grant/research support from: Amgen, Bristol-Myers Squibb, Crescendo, UCB, Dxterity, Consultant for: Amgen, Bristol-Myers Squibb, Crescendo Biosciences, UCB, AbbVie, Lilly, Pfizer, Roche  
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**AB0208 HOSPITALIZATION RATES IN PATIENTS WITH RA BY POOR PROGNOSTIC FACTORS: IMPACT OF ABATACEPT AND OTHER DISEASE-MODIFYING ANTIRHEUMATIC THERAPIES**

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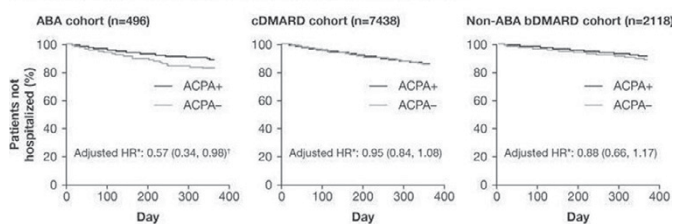
**Background:** Studies have reported that poor prognostic factors (PPF) in RA, such as high anti-citrullinated protein antibodies (ACPA), are associated with erosions, rapid radiographic progression and/or extra-articular manifestations.<sup>1,2</sup> Evidence from clinical trials and clinical practice indicate differences in treatment effects of biologic (b)DMARDs by ACPA status and/or level.<sup>3,4</sup> PPF also play an important role in clinical management of patients (pts) with RA, including inpatient admissions.

**Objectives:** To compare hospitalization rates of ACPA-positive (+) with ACPA-negative (-) pts managed with abatacept (ABA), non-ABA bDMARDs or conventional (c)DMARDs.

**Methods:** This is a retrospective cohort analysis of Clinformatics Data Mart, a database of administrative health claims including results for outpatient laboratory tests, processed by national laboratory vendors under contract with the managed care organization, for a total of ~53 million unique lives over 13 years. This analysis was restricted to adult pts (aged  $\geq 18$  years) who had at least two ICD-9-CM diagnosis codes for RA between Jan 2007 and Dec 2014 (identification period) and 12 months (M) of membership/drug benefit. Pts with ankylosing spondylitis, Crohn's disease, lupus, psoriasis or ulcerative colitis at or before the index date were excluded. ACPA+ was based on  $> 19$  or  $> 5$  U/mL, depending on the test utilized. Follow-up for pts initiating ABA was from first day of treatment to first hospitalization, end of enrolment or end of 12M follow-up. The primary outcome was all-cause hospitalization at 12M. Descriptive statistics such as Wilcoxon rank-sum test for continuous variables or Pearson's chi-square test for categorical variables were used. Cox proportional hazard model was used to examine all-cause hospitalization adjusted for age, sex, region and past hospitalization. Additional covariates included co-morbidities that were different between ACPA+/- pts. Similar analyses were performed for pts treated with cDMARDs and non-ABA bDMARDs.

**Results:** A total of 496 ABA, 7438 cDMARD and 2118 non-ABA bDMARD pts were included, with an overall past hospitalization rate of 35.5, 25.4 and 23.2%, respectively. Overall, 59.5, 41.5 and 51.7% of pts were ACPA+ in the ABA, cDMARD and non-ABA bDMARD cohorts, respectively. ACPA+ pts were older and less likely to have obstructive sleep apnoea, depression and myalgia/myositis across all cohorts. Unadjusted 12M hospitalization rate in ACPA+ vs ACPA- pts was 10.8 vs 16.4%, 13.7 vs 14.3% and 8.5 vs 10.8% in the ABA, cDMARD and non-ABA bDMARD cohorts, respectively. After controlling for baseline covariates, the hazard ratio for hospitalization in the ACPA+ (vs ACPA-) group was 0.57 (95% CI: 0.34, 0.98;  $p=0.04$ ), 0.95 (95% CI: 0.84, 1.08;  $p=0.47$ ) and 0.88 (95% CI: 0.66, 1.17;  $p=0.38$ ) in the ABA, cDMARD and non-ABA bDMARD cohorts, respectively (Figure).

Figure. Kaplan-Meier Plot for 1-Year Hospitalization According to ACPA Status



\*HR of hospitalization for ACPA+ vs ACPA- from a Cox model adjusted for age, sex, region, past hospitalization and co-morbidities.  
 $p < 0.05$ . ABA=abatacept; bDMARD=biologic DMARD; cDMARD=conventional DMARD; HR=hazard ratio

**Conclusions:** ACPA+ pts with RA treated with abatacept have a lower rate of hospitalization than ACPA- pts. This pattern was not observed with cDMARDs or non-abatacept bDMARDs. Further efforts including matching and subgroup analysis should be explored for direct comparisons between the cohorts.

**References:**

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[3] Gottenberg JE, et al. Ann Rheum Dis 2012;71:1815-9.

[4] Sokolove J, et al. Ann Rheum Dis 2016;75:709-14.

**Disclosure of Interest:** E. Alemao Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb, L. Burns Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb, Z. Guo Shareholder of: Bristol-Myers Squibb, Employee of: Bristol-Myers Squibb

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