physician visits in baseline. TCZ users were less likely to be diabetic, use methotrexate in the baseline, and to be naïve to bDMARDs. The crude incidence rate (IR) per 1000 patient-years for composite CVD among Medicare patients ranged from 13.3 (95% CI: 11.1 to 16.0) for etanercept to 19.4 (95% CI: 16.3 to 20.9) for rituximab users. The crude incidence rate for pooled TNFi users was 16.4 (15.2–17.7). Compared to TCZ, the adjusted hazard ratios were 1.03 (0.82–1.29) for abatacept, 1.25 (0.96–1.61) for rituximab, 1.13 (0.84–1.52) for etanercept, 1.33 (0.99–1.80) for adalimumab, and 1.57 (1.21–2.05) for infliximab (figure 1). There were no significant differences in CVD risk between tocilizumab and any other biologic using MarketScan data. Results were robust in numerous subgroup analyses.

Abstract OP0193 – Figure 1. Incidence rates and adjusted hazard ratios of CVD events in RA patients

Conclusions: Consistent with findings of a recently completed safety trial in RA, tocilizumab was associated with a comparable CVD risk compared to etanercept, as well as a number of other RA biologics, in two large data sources.

Disclosure of Interest: F. Xie: None declared, H. Yun Grant/research support from: BMS, E. Levitan Grant/research support from: Amgen, Consultant for: Amgen, Novartis, P. Muntner: None declared, J. Curtis Grant/research support from: AbbVie, Amgen, BMS, Corrona, Janssen, Lilly, Myriad, Pfizer, Roche/Genentech, UCB, Consultant for: AbbVie, Amgen, BMS, Corrona, Janssen, Lilly, Myriad, Pfizer, Roche/Genentech, UCB

DOI: 10.1136/annrheumdis-2018-eular.5807

OP0194 THE ASSOCIATION BETWEEN SERUM URIC ACID AND ARTERIAL STIFFNESS IN A LOW-RISK, LARGE POPULATION OF MIDDLE-AGED KOREAN

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Background: Arterial stiffness occurs because of biologic ageing and arteriosclerosis, and is most commonly measured by pulse-wave velocity. Several studies have reported that high serum uric acid may contribute to the development of a number of metabolic and haemodynamic abnormalities, and multivariate analyses in epidemiologic studies have suggested that hyperuricemia is an independent risk factor for arterial stiffness in those with comorbidities such as diabetes, hypertension, and chronic kidney disease. However, there are few reports about the association between SUA and arterial stiffness in apparently healthy populations.

Objectives: We aimed to investigate the association between serum uric acid (SUA) and arterial stiffness as evaluated by brachial ankle pulse wave velocity (baPWV) in a low-risk, large, middle-aged Korean population.

Methods: We conducted a cross-sectional study of 66,917 Koreans (38 170 men, 28 747 women) who received yearly screening with available PWV and SUA results. None of the participants had coronary heart disease, diabetes, or hypertension. SUA was divided into quintiles for assessment of its association with baPWV by multiple linear regression analysis.

Results: The average SUA level was 5.23±1.4 mg/dl, and SUA values were higher in men than in women (6.1±1.2 mg/dl vs 4.1±0.8 mg/dl). In multiple regression analysis, PWV was significantly higher in SUA quintiles 2–5 compared to the lowest group (reference) (coefficient=11.52, 18.19, 24.73, and 31.02 cm/s, respectively). In female subjects, the average difference (cm/s) of PWV between quintiles 2–5 and quintile 1 of SUA was 13.1, 22.9, 34.6, and 32.1, respectively.

Fully adjusted linear coefficient β (S.E.) was 6.62 (0.70) and 12.43 (1.33) in all participants and female subjects, respectively ($p<0.001$). In contrast, there was a J-shaped association between PWV and SUA quintile among males. When modelled continuously, each 1 mg/dl higher SUA level was associated with 0.27 higher baPWV ($p<0.001$) in the adjusted analysis.

Conclusions: These findings indicate that higher SUA levels could have an unfavourable impact on arterial stiffness as measured by baPWV in a low-risk, large, middle-aged Korean population.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.1848

OP0195 ROLE OF SEROPOSITIVITY ON MORTALITY IN RA AND THE IMPACT OF TREATMENT WITH DMARDS

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Background: Previous studies showed that RF positivity (+) in RA was associated with increased overall mortality, and that cause-specific mortality rates differed by autoantibodies. Anti-citrullinated protein antibodies (ACPAs) have been associated with cardiovascular death, and RF with death due to neoplasm and respiratory disease.¹

Objectives: To evaluate the association of serostatus (in particular, antibody [Ab] titres) with mortality and its modification by DMARDs.

Methods: Administrative claims data from Optum Clinformatics Data Mart and Humana databases (2006–2016) were used. Inclusion criteria: 2 diagnosis codes for RA plus 1 DMARD prescription; age ≥ 18 years (y); ≥ 6 months (M) baseline (BL); ± 3 M from index date). Index date was the first test date for ACPA or RF (main analysis) or the DMARD prescription date (DMARD effect on mortality analysis). Patients (pts) with ankylosing spondylitis, Crohn's disease, lupus, psoriatic arthritis or ulcerative colitis at/before index date were excluded. Based on BL Ab test, pts were categorised into Ab status of ACPA+/-, RF +/- and double +/- . Ab +pts were then categorised into 2 groups based on Ab titres. DMARD-exposed pts were categorised into biologic (b)DMARD (use of any bDMARD) and conventional (c)DMARD (use of a cDMARD but never a bDMARD) cohorts. Crude mortality rates per 1000 pt-y, as well as adjusted analysis using traditional multivariate regression and disease risk score methods, were used. Covariates were age, sex, region, physician office visits in past 3 M, indicator variable for RA diagnosis before ACPA/RF testing, past hospitalisation, medication use (steroids, NSAIDs, salicylates), DMARD use and co-morbidities.

Results: A total of 53 849 and 79 926 pts with RA had evaluable ACPA and RF status, respectively. The average (SD) age was 61.4 (15.2) and 61.8 (15.6) y in the ACPA and RF cohorts, respectively. For both ACPA and RF, mortality rates were significantly higher in Ab +vs Ab- pts, and were highest in pts with the highest Ab titres (table 1). The hazard ratios (HRs) for mortality were highest in pts with double positivity (figure 1A). HRs were higher in Ab +vs Ab- pts exposed to cDMARDs. There was no difference in mortality between Ab +vs Ab- pts in the bDMARD-exposed group (figure 1B).

Abstract OP0195 – Table 1 Crude mortality rates and HRs in patients categorised by serostatus

	Patients, n	Deaths, n	Pt-y	Crude mortality, incidence rate/1000 pt-y (95% CI)	Adjusted HR (95% CI)	P value
ACPA-	36 667	1798	1 26 451	14.2 (13.6–14.9)	1.00	
ACPA+	17 182	1276	57 719	22.1 (20.9–23.4)	1.48 (1.37–1.60)	<0.001
ACPA +Group 1*	8321	606	29 518	20.5 (18.9–22.2)	1.38 (1.25–1.52)	<0.001
ACPA +Group 2*	8861	670	28 201	23.8 (22.0–25.6)	1.60 (1.45–1.76)	<0.001
RF-	46 376	2522	1 79 247	14.1 (13.5–14.6)	1.00	
RF+	33 550	2688	1 18 583	22.7 (21.8–23.5)	1.44 (1.36–1.53)	<0.001
RF +Group 1*	16 758	1098	60 393	18.2 (17.1–19.3)	1.18 (1.09–1.27)	<0.001
RF +Group 2*	16 792	1590	58 190	27.3 (26.0–28.7)	1.79 (1.51–2.11)	<0.001

*Based on antibody titre: Group 1=lower titre; Group 2=higher titre