Ingelheim, Chugai, Lilly, MSD, Pfizer, Roche, Sanofi-Aventis, UCB, A. Strangfeld Speakers bureau: BMS, MSD, Pfizer, Roche, Sanofi-Aventis, A. Zink Speakers bureau: AbbVie, BMS, MSD, Pfizer, Roche, UCB
DOI: 10.1136/annrheumdis-2017-eular.4697

SATURDAY, 17 JUNE 2017

Rheumatoid arthritis - comorbidity and clinical aspects

SAT0088 HDL CHOLESTEROL EFFLUX CAPACITY IN RHEUMATOID ARTHRITIS PATIENTS: CONTRIBUTING FACTORS AND RELATIONSHIP WITH SUBCLINICAL ATHEROSCLEROSIS

B. Tejera Segura[1], M. Macía-Díaz[2], J.D.M. Machado[2], A. de Vera-González[3], A. González-Delgado[3], J.M. Olmos[4], J.L. Hernández[4], F. Díaz-González[1], M.A. González-Gay[5], I. Ferraz-Amaro[1]. *[1]Division of Rheumatology; [2]Pharmacology Department; [3]Central Laboratory Division, Hospital Universitario de Canarias, Tenerife; [4]Division of Internal Medicine; [5]Rheumatology Department, Hospital Universitario Marqués de Valdecilla-IDIVAL, Santander, Spain*

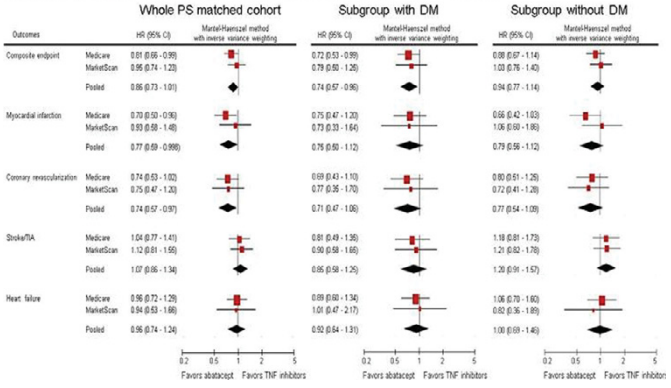
Background: Lipid profiles appear to be altered in rheumatoid arthritis (RA) patients due to disease activity and inflammation. Cholesterol efflux capacity (CEC) has not only been linked to cardiovascular events in the general population, but also to be impaired in RA patients.
Objectives: To analyze whether CEC is related to subclinical atherosclerosis, as determined by the presence of carotid plaque or increased levels of carotid intima-media thickness (cIMT) in RA patients. Secondarily, we aimed to describe the disease-contributing factors that are related to CEC as an expression of the abnormalities in the lipid profile associated with the disease.
Methods: Cross-sectional study that encompassed 401 individuals; 178 patients with RA and 223 sex-matched controls. CEC was measured using an *in vitro* assay, lipoproteins serum concentrations, and standard lipid profile were assessed in patients and controls. Carotid intima-media thickness and carotid plaques were assessed in RA patients. A multivariable analysis was performed to evaluate the relation of CEC with RA-related data, lipid profile and subclinical carotid atherosclerosis.
Results: Mean CEC was not significantly different between RA patients (18.9 ± SD 9.0%) and controls (16.9±10.4%), p=0.11. Demographic variables were not associated with CEC except for a correlation with male gender that was only found in RA patients, but not in controls. Systolic blood pressure inversely correlated with CEC in controls (beta coefficient -0.1 [-0.2–0.0] %, p=0.025). In RA patients, a similar trend was found although a statistically significant difference was not reached. Neither the traditional cardiovascular risk factors nor the cardiovascular co-morbidity-related data were associated with CEC. Similarly, lipid profile did not show any relationship with CEC in patients or controls. ESR tended to be associated with a lower CEC although it did not reach statistical significance. RA patients with low (beta coef. -5.2 [-10.0–0.3] %, p=0.039) and moderate disease activity (beta coef. -4.6 [-8.5–0.7] %, p=0.020) were associated with inferior levels of CEC when compared to patients in remission. CEC was not found to be associated with cIMT in RA patients. However, higher CEC was associated with a protective effect for the presence of carotid plaque in RA patients. This relationship was maintained even after multivariate analysis (OR 0.94 [0.89–0.98], p=0.015).
Conclusions: Our study, which includes the largest series of RA patients ever assessed for CEC, reveals for the first time that CEC is related to subclinical atherosclerosis in RA patients. The fact that CEC is also associated with disease activity reinforces the idea that CEC may be a mediator between disease activity and subclinical atherosclerosis.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.4100

SAT0089 COMPARATIVE CARDIOVASCULAR SAFETY OF ABATACEPT AND TUMOR NECROSIS FACTOR INHIBITORS IN RHEUMATOID ARTHRITIS PATIENTS WITH AND WITHOUT TYPE 2 DIABETES: A POPULATION-BASED COHORT STUDY

E.H. Kang[1,2], Y.P. Jin[1], G. Brill[1], J.P. Lewey[1,3], E.P. Patorno[1], R. Desai[1], S.C. Kim[1]. *[1]Division of Pharmacoepidemiology and Pharmacoeconomics, Harvard Medical School and Brigham and Women's Hospital, Boston, United States; [2]Division of Rheumatology Department of Internal Medicine, Seoul National University Bundang Hospital, Seongnam, Korea, Republic Of; [3]University of Pennsylvania, Philadelphia, United States*

Background: Patients with rheumatoid arthritis (RA) are at high risk of developing cardiovascular disease (CVD) and may benefit from potent disease-modifying anti-inflammatory drugs such as biologics. Since diabetes mellitus (DM) is a major risk factor for CVD, RA patients with DM constitute a high CVD risk subgroup, calling for particular attention. However, there is a lack of knowledge on the comparative cardiovascular safety of different biologics in RA patients with DM.
Objectives: To examine the comparative cardiovascular safety of abatacept versus TNF inhibitors in RA patients with and without DM.
Methods: RA patients enrolled in both public (Medicare) and commercial (Truven MarketScan) health plans in the U.S. who newly initiated abatacept or TNF inhibitors were eligible. The primary outcome of interest was a composite CVD endpoint of myocardial infarction (MI), stroke/transient ischemic attack (TIA), and coronary revascularization. The secondary outcomes included each component of the composite CVD endpoint and heart failure (HF). After 1:1 propensity score (PS) matching between two exposure groups, Cox proportional hazard model was used to estimate the hazard ratio (HR) and 95% confidence interval (CI) for each outcome, comparing abatacept to TNF inhibitors. PS matching was separately done in subgroups with or without baseline DM in each database. PS-matched HRs from the two databases were pooled using an inverse variance–weighted fixed-effect model.
Results: We identified 31,899 Medicare enrollees (6,107 new users of abatacept and 25,792 new users of TNF inhibitors) and 71,956 commercial enrollees (6,942 new users of abatacept and 65,464 new users of TNF inhibitors) with RA. After PS matching, 2,119 pairs with DM and 3,984 pairs without DM were identified in Medicare and 1,371 pairs with DM and 5565 pairs without DM in MarketScan. The PS matched HR (95% CI) for the composite CVD endpoint in the whole cohort was 0.81 (0.66–0.99) in Medicare and 0.95 (0.74–1.23) in MarketScan with a pooled HR (95% CI) of 0.86 (0.73–1.01). In the subgroup with DM, HR (95% CI) for composite CVD endpoint was 0.72 (0.53–0.99) in Medicare and 0.79 (0.50–1.25) in MarketScan with a pooled HR (95% CI) of 0.74 (0.57–0.96). In the subgroup without DM, HR (95% CI) was 0.88 (0.67–1.14) in Medicare and 1.03 (0.76–1.40) in MarketScan with a pooled HR (95% CI) of 0.94 (0.77–1.14). For secondary outcomes (Figure 1), the pooled HR (95% CI) was 0.77 (0.59–1.00) for MI and 0.74 (0.57–0.97) for coronary revascularization in the whole cohort. There was a trend toward a decreased risk, albeit statistically insignificant, for MI and coronary revascularization in each subgroup. There was no significant difference in the risk for stroke/TIA and HF.

Figure 1. Cardiovascular safety of abatacept compared to TNF inhibitors



Conclusions: Among RA patients, abatacept may be associated with a reduced risk of coronary events compared to TNF inhibitors, particularly in patients with DM. The risk of HF was not different between these two groups.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.2407

SAT0090 SUCCESS RATE OF BLOOD PRESSURE GOAL ACHIEVEMENT IN INFLAMMATORY JOINT DISEASES

S. Rollefstad, P. Norheim, E. Ikdahl, G. Wibetoe, A.G. Semb. *Preventive Cardio-Rheuma Clinic, Department of Rheumatology, Diakonhjemmet Hospital, Oslo, Norway*

Background: The excess risk of cardiovascular disease (CVD) in patients with inflammatory joint diseases (IJD) is attributable to several risk factors, including a high prevalence of hypertension. However, there is limited knowledge on the effect of antihypertensive treatment (a-HTT) in these patients.
Objectives: Our objective was to initiate a-HTT when indicated and treat to guideline recommended blood pressure (BP) goal in IJD patients. We also aimed to evaluate the effect of a-HTT in this patient population, and which factors were associated with BP goal attainment.
Methods: Patients with IJD (n=765) were referred from a rheumatology outpatient clinic or general practitioners to a preventive cardio-rheuma clinic. All patients underwent a CVD risk evaluation, including BP measurements (performed using and Omron M7 apparatus). Antihypertensive treatment was initiated in accordance with guidelines, and the BP treatment goal was <140/90 mmHg.
Results: Of the 765 IJD patients referred (rheumatoid arthritis n=450, ankylosing spondylitis n=210 and psoriatic arthritis n=105), 104 (13.6%) had an indication for BP lowering, while 224 (29.3%) were already using a-HTT at the first consultation. For those where a-HTT was initiated at baseline (n=104), there was a highly significant change in BP from first to final consultation (Fig 1a). BP goal was achieved in 84 (80.8%) patients (Fig 1b), using mean±SD 3.1±1.7 consultations. Dose adjustments was done in 38 (36.5%) of the patients with median (IQR) a-HTT dose adjustments of 1 (1, 1.25). In 9 (8.7%) patients the