a-HTT was changed. Systolic BP ($p < 0.0001$) was significantly associated with BP goal attainment in age- and sex adjusted logistic regression analyses, while the use of anti-rheumatic medication or inflammatory biomarkers at baseline was not. Patients with the lowest systolic BP were more likely to achieve BP goals. For patients already on a-HTT ($n=224$), only 52.7% had a BP <140/90 mmHg at baseline. After up titration or change of a-HTT, the percentage of patients achieving BP goal in this group increased to 82.6%.

Conclusions: This is to our knowledge the first prospective report on success rate of BP goal achievement in patients with IJD. Approximately 80% reached BP target, which is even a higher proportion than what is shown in the general population. Treatment to BP goal is feasible in patients with IJD, and is not complicated by inflammation or use of anti-rheumatic medication.

Disclosure of Interest: None declared

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SAT0091 IS CLINICAL ARTHRITIS ALWAYS PRECEDED BY SUBCLINICAL INFLAMMATION? A LONGITUDINAL STUDY AT JOINT LEVEL IN PATIENTS WITH ARTHRALGIA THAT DEVELOPED ARTHRITIS

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Background: The clinical phase of Rheumatoid arthritis (RA) is preceded by a phase with subclinical inflammation. MRI can detect subclinical inflammation and, at patient level, this is predictive for the development of clinical arthritis. However, at joint level it is unknown how arthritis develops. It is unknown how frequently joints with subclinical inflammation progress to clinical arthritis, and vice versa, how often joints that developed clinical arthritis had local subclinical inflammation during the preceding phase of arthralgia. A longitudinal MRI study in patients that developed arthritis can unravel if arthritis development is restricted to some locations in which the severity of inflammation increases over time or, alternatively, if the process is more generalized with a weak association between the locations of subclinical inflammation and subsequent clinical arthritis.

Objectives: This longitudinal study at joint level during progression from pre-RA to RA determined the relation between the location of subclinical inflammation and clinical arthritis over time.

Methods: 290 small joints (4 MCPs, 1 wrist, 5 MTPs per person) of 29 patients that presented with arthralgia and developed clinical arthritis were studied with 1.5T MRI at both time-points. MRIs were evaluated for BME, synovitis and tenosynovitis by three readers (ICCs 0.98, 0.96 and 0.97) that were blind to clinical data and the order in time. Subclinical inflammations was defined as presence of BME, synovitis and/or tenosynovitis.

Results: The median time between presentation with arthralgia and clinical arthritis development was 17 weeks. At presentation with arthralgia 68 joints had subclinical inflammation and no significant association was found between joint tenderness and the presence of local MRI-detected subclinical inflammation (OR 0.98; 95% CI 0.48–1.9). Over time, 21% of 68 joints had resolution of subclinical inflammation, 60% had persistent subclinical inflammation and 19% developed

clinical arthritis. At arthritis development 37 joints were swollen. Of these, 24 (65%) had no prior subclinical inflammation at the time of presentation with arthralgia (Figure).

Conclusions: This first longitudinal MRI-study on joint level in pre-RA suggested that the majority of joints that developed clinical arthritis had no (long-lasting) preceding phase with subclinical inflammation.

Disclosure of Interest: None declared

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SAT0092 PROGRESSION OF ATHEROSCLEROSIS OVER 5 YEARS IN A COHORT OF EARLY INFLAMMATORY ARTHRITIS PATIENTS: RESULTS FROM THE NORFOLK ARTHRITIS REGISTER

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Background: Carotid artery intimal medial thickness (IMT) on ultrasound (US) is a validated measure of subclinical cardiovascular (CV) disease. Rates of IMT progression of 0.001cm/year¹ have been observed in the general population and faster progression is associated with increased CV risk.

Objectives: To describe change in IMT over 5 years in a cohort of early inflammatory arthritis (IA) patients and the association of IMT with estimated CV risk and IA related factors.

Methods: Between 2004 and 2010, patients enrolling in the Norfolk Arthritis Register (a UK inception cohort of IA patients) aged from 16 to 65 years with symptom duration less than 2 years, were invited to take part in the NOAR CVD sub-study. In addition to standard IA disease assessments at 0, 2 and 5 years, traditional CV risk factors were measured at baseline and Framingham based 10-year CV risk score was calculated. IMT was measured in the common carotid arteries at baseline and year 5 using a previously validated US protocol. Patient reported clinical CV events at follow up visits which were then verified by a physician. The associations of baseline CV risk and IA characteristics (tender and swollen joint count, rheumatoid factor, CRP, health assessment questionnaire (HAQ), baseline disease modifying anti-rheumatic therapy use) with baseline IMT (IMT₀) and change in IMT at year 5 (IMT_Δ) were tested using non-parametric statistics. The association of IMT_Δ with IA characteristics over time (cumulative joint counts, change in HAQ) and medication use during follow up (statins, biologic therapy) with IMT_Δ were explored using non-parametric statistics.

Results: 201 patients with a median (IQR) age and symptom duration of 51 (42, 58) years and 10.4 (7.7, 14.4) months respectively were studied. 143 (71%) subjects were female. Median IMT₀ was 0.06 (0.05, 0.07) cm and median IMT_Δ at 5-year follow up period was 0.01 (0, 0.01) cm with an estimated median annual IMT_Δ of 0.002cm. IMT had progressed in 104 (51.5%) patients and regressed in 23 (11.4%) patients by year 5. The median CV risk score at baseline was 5.6 (2.6, 10.7)% and was associated with IMT₀ but not IMT_Δ ($r=0.46, p<0.01$; $r=0.03, p=0.73$ respectively). There was no association between baseline IA characteristics and IMT₀ or IMT_Δ. There was a trend towards an association between cumulative swollen joint count and IMT_Δ ($r=0.14, p=0.057$) but no association with cumulative tender joint count, change in HAQ, statin or biologic use (all $p>0.05$). CV events occurred in 5 patients during follow up. Of these patients, 3 had IMT progression, 1 had no change and 1 had IMT regression.

Conclusions: Overall, patients with early IA had a higher than expected rate of IMT progression; a significant proportion however had regression in IMT over time. Trajectories of CV risk may vary within the IA population and identifying protective factors will help to better target strategies for CVD prevention in IA.

References:

[1] G Howard et al. Carotid artery intimal-medial thickness distribution in general populations as evaluated by B-mode ultrasound. ARIC Investigators. Stroke. 1993;24:1297–1304.

Disclosure of Interest: None declared

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SAT0093 HIGH TRIGLYCERIDES AND LOW HDL LIPID PROFILE AS A SURROGATE MARKER OF HDL DYSFUNCTION IN RA: POTENTIAL LINKS WITH INFLAMMATION AND OXIDATIVE STATUS

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Background: the interactions between inflammation and lipid profile in RA are

poorly understood. The study of lipid profiles in RA has been biased towards lipoprotein levels, whereas those of triglycerides (TG) and lipoprotein functionality have been neglected.

Objectives: since recent findings point to an emerging role for TG and TG-rich lipoproteins (TRL) on inflammation, we aimed to evaluate a combined lipid profile characterized by high TG and low HDL levels (TG^{high}HDL^{low}) in RA.

Methods: lipid profiles were analyzed in 113 RA patients, 113 healthy controls (HC) and 27 dyslipemic subjects (DL). A group of 13 biological-naïve RA patients was prospectively followed for 3 months upon TNF α -blockade. Serum levels of inflammatory mediators were assessed by immunoassays. PON1 activity and Total Antioxidant Capacity (TAC) were quantified in serum. PON1 rs662 status was evaluated by RT-PCR

Results: the prevalence of the TG^{high}HDL^{low} profile was similar among RA patients (29/113), HC (30/113) and DL (11/27), linked to higher TRL levels in all groups. However, this profile was associated with increased CRP (p=0.012), TNF α (p=0.004), MCP-1 (p=0.004), IP-10 (p=0.018) and leptin (p<0.001) serum levels in RA, where decreased PON1 activity and TAC were found (both p<0.001). TRL serum levels were positively correlated to inflammatory mediators, whereas a negative association was found for PON1 activity (r=-0.203, p=0.036). These findings remain after excluding patients with previous CV events or those under statins. No associations were observed in the HC and DL groups. When RA patients were stratified by PON1 rs662 status, these associations were restricted to the low activity genotype (QQ) (TNF α : p=0.002, MCP-1: p=0.013, EGF: p=0.047, IP-10: p=0.018 and leptin: p=0.002), whereas no effect of the lipid profile was observed in those harboring the QR or RR genotypes (all p>0.050). As expected, QQ-patients exhibited a lower PON1 activity compared to the other genetic variants (both p<0.010). The TG^{high}HDL^{low} prevalence was related to a decreased anti-TNF α usage in the cross-sectional sample (p=0.004). A poor clinical response upon TNF α -blockade was related to an increasing prevalence of the TG^{high}HDL^{low} profile over treatment (p=0.021) and elevated baseline TRL levels (p=0.042).

Conclusions: the TG^{high}HDL^{low} profile is associated with systemic inflammation, increased TRL levels, decreased PON1 activity and a poor clinical outcome upon TNF α -blockade in RA. Overall, these findings support the link between inflammation and lipid profile, oxidative status and TRL having a pivotal role. The TG^{high}HDL^{low} profile can be proposed as a surrogate marker of HDL dysfunction in RA.

Disclosure of Interest: None declared

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SAT0094 METABOLIC AND CARDIO-VASCULAR BENEFITS OF HYDROXYCHLOROQUINE IN PATIENTS WITH RHEUMATOID ARTHRITIS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: Cardiovascular disease (CVD) is the leading cause of mortality in rheumatoid arthritis (RA) patients (1). Hydroxychloroquine (HCQ) has been shown to improve major outcomes like survival rates in other inflammatory diseases, like systemic lupus (2).

Objectives: The aim of our study was to assess currently available literature on the cardiovascular impact of hydroxychloroquine (HCQ) in patients with RA.

Methods: We systematically searched literature (via PubMed, Embase and abstracts from recent ACR and EULAR congresses) for studies evaluating the effects of HCQ, whether in monotherapy or in combination with other conventional synthetic disease modifying antirheumatic drugs (csDMARDs) on cardiovascular outcomes or known risk factors for CVD in RA patients (lipid profiles, diabetes incidence, insulin resistance and incidence of CVD). A meta-analysis was performed with Review Manager Software, with random effects models, whenever methodologically possible and relevant. Data were extracted by one investigator and independently checked by another.

Results: The literature search revealed 185 articles and abstracts of potential interest, and further examination resulted in 16 studies fulfilling required criteria for preplanned analyses regarding the cardiovascular impact of HCQ in RA. For lipid profiles, the mean difference (mg/dL) between HCQ users versus nonusers was -9.82 (95% confidence interval [95% CI] 14.03; 5.60) for total cholesterol, -10.61 [14.17; 7.04] for low density lipoprotein, -19.15 [27.20; 11.10] for triglycerides and +4.13 [2.22; 6.04] for high density lipoprotein (figure 1); with respectively a decrease (mg/dL) of 13.15 [20.96; 5.34], 12.35 [20.14; 4.36], 12.54 [28.94; 3.86] and an increase of 1.67 [0.96; 4.31] after HCQ initiation. Diabetes incidence was reduced in "HCQ ever users" versus "patients who never used HCQ" with a hazard ratio of 0.59 [0.49; 0.70]. In addition, HCQ seems to decrease insulin resistance and incidence of cardiovascular events but data were too scarce for meta-analysis.

Conclusions: Beside its limited efficacy on disease activity, this study supports the interest of HCQ on lipid profiles, and diabetes incidence, and to a lesser extent on cardio-vascular events and insulin resistance in RA patients. Therefore, this study suggests that HCQ may be of some interest in RA, in combination with other csDMARDs.

References:

[1] Aviña-Zubieta JA, Choi HK, Sadatsafavi M, Etminan M, Esdaile JM, Lacaille D.

Figure 1. Forest plot for the mean difference (mg/dL) between HCQ users and non-users of total-cholesterol (A), low-density-lipoprotein (B), high-density-lipoprotein (C), and triglycerides (D).

Risk of cardiovascular mortality in patients with rheumatoid arthritis: a meta-analysis of observational studies. *Arthritis Rheum.* 2008 Dec 15;59(12):1690-7.

[2] Petri M. Hydroxychloroquine use in the Baltimore Lupus Cohort: effects on lipids, glucose and thrombosis. *Lupus.* 1996 Jun;5 Suppl 1:S16-22.

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SAT0095 INCRETINS-INSULIN AXIS IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) patients have higher levels of resistance to the action of insulin (IR) compared to healthy subjects. The "Incretin effect" consists in a greater release of insulin by the pancreas when there is a gastrointestinal glucose stimulation, compared to an intravenous stimulation. It is known that this effect is altered in patients with IR.

Objectives: To determine if the incretins-insulin axis is altered in patients with RA and if this correlates to IR in patients with RA, as well as if it is explainable by certain features of the disease.

Methods: Cross-sectional study that includes 361 non-diabetic individuals, 151 patients with RA and 210 controls. Serum levels of insulin, C-peptide, Amylin, glucagon-like peptide 1 (GLP-1), gastric inhibitory polypeptide (GIP) and dipeptidyl peptidase 4 (DPP-4) were analyzed in patients and controls. Indexes of resistance and sensitivity to insulin activity were determined by HOMA2.

A "meal test" consisting on the intake of 500 kcal was performed to a subset of 10 patients and 10 controls, in order to determine the postprandial curves of glucose, insulin, C-peptide, GLP-1 and GIP. Differences between patients and controls as well as the relationship of selected characteristics of the disease with the baseline and postprandial levels of incretins were analyzed using multivariate regression. During the meal test, areas under the curve (AUC), maximum concentrations and the minutes of response were compared between patients and controls. This study was approved by the Clinical Trial Committee of the University Hospital of the Canary Islands.

Results: Patients with RA showed, at baseline and after multivariate adjustment, higher levels of insulin (9.8±6.5 vs 13.0±13.4 U/ml, p=0.05), C-peptide (1.53±0.77 vs. 3.37±2.94 ng/ml, p=0.00), GLP-1 (0.49±1.28 vs. 0.71±0.22, p=0.00) and GIP (0.37±0.40 vs. 1.78±0.51, p=0.00). Similarly, IR indexes such as HOMA2-IR and HOMA2%B, built with insulin or C-peptide, were higher in patients with RA compared to controls. Patients with RA, showed significantly lower levels of DPP-4 (811±459 vs. 696±301 ng/ml, p=0.02) after multivariate analysis. Amylin levels did not differ between patients and controls. Both C-reactive protein (beta coefficient 0.54, 95% CI 0.16–0.96, p=0.013) and Erythrocyte Sedimentation Rate (beta coef. 0.01, 0.00–0.01, p=0.033) showed a positive relationship with HOMA2%B and GIP levels, respectively. The presence of anti-cyclic citrullinated peptide antibody (beta coef. 157 CI 58–256, p=0.002) and the levels of disease activity measured by DAS28 (beta coef. 46, CI 6–87, p=0.026) and CDAI (beta coef. 1.27 (0.28–2.26), p=0.012) showed a positive relationship with the levels of DPP-4. Patients with RA showed Pearson correlation coefficient of incretins, DPP-4 with insulin, C peptide and IR lower compared to control group. After the meal test patients with