Methods: Patients with RA from two teaching hospitals, participating in a prospective observational study completed the HAQ-DI and EQ5D-5L at 0–6-12 month follow-up visits. Inclusion criteria: ACR RA diagnosed patients, on biologic treatment and whose disease activity remained stable at least for 3 months EQ5D-5L is a standardized, generic instrument for describing and valuing health and QoL, consisting in a five-dimensional descriptive system (mobility, self-care, usual activities, pain/discomfort and anxiety/depression) and a visual analogue scale. A country-specific tariff converts patient's answer to a 0–1 (full health) utility index. HAQ-DI is a self-completed questionnaire used to assess the functional ability using 20 items, distributed across 8 dimensions and resulting in a four-level disability scale (0–3). In addition, socio-demographic and clinical data where recorded.To estimate the EQ5D-5L utility index OLS models were built. As this index is bounded to the [-0.416, 1] interval, Tobit models were also considered. Hereafter, the index was transformed to the open interval (0,1) and estimated through beta regression with a logit link. To determine the relationship grade between the index and the HAQ-DI scale and obtain residuals without trend, GAM models were used. Best fitting models were determined by AIC, MAE and RMSE. All analyses were performed using R software

Results: 217 questionnaires fulfilled by 77 patients. Mean (SD) age was 57.0 (12.9), 87% women, AR duration 13.7 (7.1), mean DAS28 2.72 (1.00) and HAQ-DI 0.77 (0.60). Baseline EQ5D index: 0.768 (0.182). All the OLS estimation models resulted in the interval limits defined by the index, so Tobit models were not considered. When considering the linear model we obtained the best results with the HAQ-DI term and its third power: EQ5D5L = 0.9232 – 0.1760×HAQ – 0.0172×HAQ^3 (AIC=–221.62; MAE=0.0974; RMSE=0.1363); for beta regression, we obtained the best model with the HAQ-DI to the first power alone: logit (EQ_01) = 2.5821 – 1.1165×HAQ (AIC=–444.4; MAE=0.0691; RMSE=0.0958). Considering the AIC and the residuals together, we obtained the best fitting model with the beta regression approach, with neither age nor sex

Conclusions: So far, only a utility function using HAQ-DI and an older EQ5D-3L version was available for Spain (Carreño,2011). This updated utility function can be used as a practical approach to predict RA patients' QoL and EQ5D utility score for Spain when clinicians/researchers need them for clinical practice or cost-effectiveness analyses and generic QoL measurements are not available

Disclosure of Interest: None declared

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THU0110 THE IMPORTANCE OF SUSTAINED REMISSION FOR LONGTERM OUTCOMES IN PATIENTS WITH RHEUMATOID ARTHRITIS

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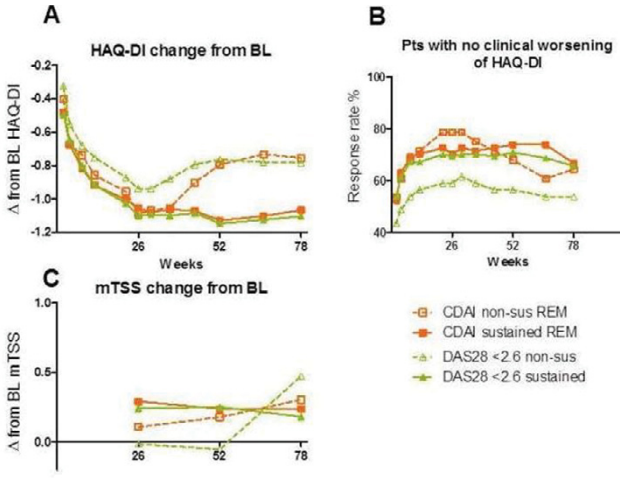
Background: In patients (pts) with rheumatoid arthritis (RA), the long-term impact of sustained versus (vs) transient clinical remission (REM) has not been assessed thoroughly, although REM duration has been shown to affect structural outcomes[1]. The relationship of different definitions of clinical remission (REM) with function and structural integrity has not been assessed.

Objectives: To explore the importance of sustained REM or disease control for long-term outcomes, and assess various definitions of REM in adalimumab (ADA) long-term trials.

Methods: Data are from 2 trials of ADA in early RA pts; In PREMIER, pts received ADA, methotrexate (MTX) or ADA+MTX for 2 years (yrs), after which they could enter an open label (OL) period for upto 8 yrs[2]. In OPTIMA, pts received ADA+MTX, or placebo (PBO) +MTX for 26 weeks (wks). Based on whether or not pts achieved DAS28-CRP <3.2 at wks 22 and 26, pts withdrew ADA, continued on PBO+MTX, ADA+MTX or OL ADA+MTX until Wk 78[3]. For this analysis, non-sustained REM/disease control was defined as meeting one of the following at 6 months but not 1 yr: DAS28-CRP <2.6; simplified disease activity index (SDAI) ≤3.3; clinical disease activity index (CDAI) ≤2.8. Sustained REM/disease control was defined as meeting these criteria at both 6 months and 1 yr. The mean change from baseline in health assessment questionnaire-disability index (ΔHAQ-DI), or modified total Sharp score (ΔmTSS), and the number of pts without clinical worsening of HAQ-DI (Δ≤0.22) were assessed over 78 wks for OPTIMA, or 5 yrs for PREMIER. NRI and LOCF were used for binary and continuous variables, respectively.

Results: In OPTIMA, by any of the REM criteria, pts in sustained REM had larger mean ΔHAQ-DI over time (Fig 1A) vs pts in non-sustained REM. Pts with non-sustained DAS28-CRP <2.6 vs non-sustained CDAI REM had numerically smaller ΔHAQ-DI up to Wk 52. Over time, more pts in sustained vs non-sustained REM using DAS28-CRP<2.6 (but not CDAI or SDAI criteria) did not have clinical worsening of HAQ-DI, possibly due to more suppression of inflammatory components upon achieving CDAI REM but not DAS28-CRP <2.6 in these early RA pts (Fig 1B). At Wk 78, ΔmTSS at Wk 78 was smaller for pts in sustained vs non-sustained DAS28-CRP <2.6, and similar for sustained and non-sustained CDAI REM (Fig 1C). Somewhat fewer pts at Wk 78 may have contributed to some variability. Trends were similar in PREMIER (not shown).

Conclusions: Pts who were in sustained disease control/REM had better clinical, functional and radiographic outcomes over the long- term, vs pts in a more transient state, regardless of the REM criteria used, although for CDAI REM, functional and radiographic outcomes were similar for sustained and non-sustained REM, in line with its higher stringency.



For LOCF: CDAI non-sustained REM N=28, CDAI sustained REM N=84, DAS28 <2.6 non-sustained, N=39, DAS28 <2.6 sustained, N=137; For NRI: CDAI non-sustained REM at Wk 26, N=22, at Wk 78, N=18; CDAI sustained REM at Wk 26, N=59, at Wk 78, N=56 DAS28 <2.6 non-sustained, at Wk 26, N=23, at Wk 78, N=21; DAS28 <2.6 sustained, at Wk 26, N=95, at Wk 78, N=90

References:
[1] Aletaha et al, Arthritis & Rheum. 2009;5:1242.
[2] Breedveld et al. 2006. Arthritis & Rheum;54:26.
[3] Smolen et al. 2014. Lancet;383:321.

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THU0111 NOVEL AUTOANTIBODY PROFILES IN RHEUMATOID ARTHRITIS AND THEIR ASSOCIATION WITH RADIOGRAPHIC PROGRESSION IN THE SCOTTISH EARLY RHEUMATOID ARTHRITIS INCEPTION COHORT AND BIOBANK (SERA)

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Background: Antibodies against citrullinated peptides are useful in the diagnosis of rheumatoid arthritis (RA) and are associated with poorer prognosis and greater radiographic progression. Novel autoantibodies recognising several post-translational modifications (PTM) are now emerging including anti-carbamyl, and anti-acetyl antibody classes. Less is known of their prognostic significance.

Objectives: To determine the prevalence of autoantibodies to modified vimentin and/or CCP2 (anti citrullinated (CCP), anti carbamylated (Carb), anti acetylated (Acet)) in a subset of the Scottish Early RA (SERA) inception cohort (n=212) and to correlate them with baseline and radiographic progression over 12 months.

Methods: Baseline and 12m hand and foot radiographs were scored by two readers independently, blinded to patient information and time order according to the Sharp van der Heijde score (SvH). Serum samples from the SERA biobank were analysed with recently published anti-modified protein assays. Patients with citrullinated antibodies (CCP) had antibodies to modified vimentin and/or antibodies to CCP2. Clinical, radiographic and autoantibody data were analysed in R.

Results: See Table 1.

In patients with early RA four main antibody profiles were detected: patients