Methods: One-hundred-and-forty-three patients with sustained disease activity score (DAS28-CRP)>2.6 and no radiographic progression the previous year were included. bDMARD was reduced to 2/3 of standard dose at baseline, ½ after 16 weeks, and discontinued after 32 weeks. Patients who flared (defined as either DAS28-CRP>2.6 and DAS28-CRP>1.2 from baseline, or erosive progression on X-ray and/or MRI) stopped tapering and were escalated to the previous dose level.

Results: One-hundred-and-forty-one patients completed 2 year follow-up. At 2 years, 87 patients (62%) had successfully tapered bDMARDs, with 26 (18%) receiving 2/3 of standard dose, 39 (28%) ½ dose and 22 (16%) having discontinued; 54 patients (38%) were receiving full dose. DAS28-CRP0.4 (median [interquartile range]) and mean Total-Sharp-Score2yrs was 0.01 (1.15) (mean[SD]). Radiographic progression was observed in 9 patients (7%). Successful tapering was independently predicted by: <1 previous bDMARD, male gender, low baseline MRI combined inflammation score and low MRI combined damage score. Negative IgM-rheumatoid factor predicted successful discontinuation. The association between potential predictors and the proportion of patients with successful tapering of bDMARDs is shown in figure 1.

Conclusions: By implementing a clinical guideline, 62% of RA patients in sustained remission in routine care were successfully tapered, including 16% successfully discontinued at 2 years. Radiographic progression was rare. IgM-RF was an independent predictor for successful discontinuation of bDMARDs. Maximum one bDMARDs, male gender, and low baseline MRI combined inflammation and MRI combined damage scores were independent predictors for successful tapering.

Disclosure of Interest: None declared


SYNOVIAL CELL INFILTRATION IN ACPA-VE PATIENTS DISPLAYS SIMILAR SIGNS TO OTHER SERONEGATIVE INFLAMMATORY ARTHRITIS, RESULTS FROM THE PATHOBIOLOGY OF EARLY ARTHRITIS PHOTON (PEACH)

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Background: There is increasing evidence to suggest that ACPA +ve and ACPA-ve RA are distinct diseases. Current data demonstrates overlap in classification criteria between ACPA-ve RA and other sero negative inflammatory arthritides such as PsA. Associated with this is a variable prognosis and response to treatment for patients with ACPA-ve RA. Biomarkers capable of refining diagnosis and improving on current classification criteria early in the disease course for patients with ACPA-ve RA are thus urgently needed. Data examining the synovial pathophysiological relationship between PsA and ACPA-sRA is currently limited although has the potential to identify different specific synovial cellular and molecular signatures.

Objectives: Therefore, the aim of this study is to examine in a cohort of therapy naïve, early inflammatory arthritic patients, whether ACPA-ve RA can be defined at disease initiation according to synovial pathological architecture.

Methods: A total of 186 consecutive DMARD naïve inflammatory arthritides patients (disease duration <1 year) recruited as part of the multicentre PEACH study at Barts Health NHS Trust were evaluated. All patients underwent a baseline synovial biopsy of a clinically active joint along with collection of inflammatory markers (CRP). Following H and E staining, sections underwent immunohistochemical staining and semi-quantitative scoring (0–4) to determine the degree of CD20+ B cells, CD3+ T cells, CD68+ low and sublining (I) and sublining (II) macrophage and CD138+ plasma cell infiltration. Sections were categorised into three pathotypes: (i) Fibro(G)+CD68 SL ≥2 and or CD3, CD20, CD138 <1, (ii) Myeloid(M)+CD68SL ≥2, CD20 <1 and or CD3 >1 and (iii) Lymphoid(L)+grade 2–3 CD20 + aggregates, CD20 ≥2.

Results: 90/186 patients were classified as ACPA+ve RA. 55/186 as ACPA-ve RA and 41/186 as PsA. 80% of synovial samples were collected from small joints (wrist, MCP,PIP). All 186 samples were suitable for analysis. Results confirmed that C-reactive protein (CRP) as inflammatory marker does not differentiate between subgroups (p=0.41). Significantly higher degree of immune cell infiltration was seen between ACPA+ve vs ACPA-ve and ACPA+ve vs PsA but not between ACPA-ve and PsA (figure 1). When grouping patient between clinical subgroups...
Conclusions: Our results suggest that the synovial cell infiltrate (B cells, T cells, macrophages and plasma cells) in ACPA-ve RA is significantly different from ACPA +ve patients. They also suggest shared pathophysiological mechanisms between PsA and ACPA-ve RA and support a role for further refinement of diagnosis of ACPA-ve RA according to synovial pathology.

Disclosure of Interest: None declared


DOES TREATMENT STRATEGY INFLUENCE THE ABILITY TO ACHIEVE AND SUSTAIN DMARD-FREE REMISSION IN RA? RESULTS OF A LINGUODINICAL STUDY COMPARING AN INTENSIVE DAS-STEERED TREATMENT STRATEGY WITH TREAT-TO-TARGET IN ROUTINE CARE

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Background: Disease-modifying anti-rheumatic drug (DMARD)-free remission is an achievable outcome in rheumatoid arthritis (RA). The influence of treatment strategy on the ability to achieve and sustain this outcome is unclear. Therefore, we compared the prevalence and sustenance of DMARD-free remission in RA-patients treated in a trial with intensive DAS-steered care aimed at DMARD-free remission versus RA-patients treated in routine care.

Methods: 279 consecutive RA-patients (2010 criteria), diagnosed in the Leiden University Medical Centre between March 2007-September 2010, were studied. Of these, 155 participated in a DAS <1.6 steered trial aimed at DMARD-free remission (IMPROVED-study). These patients were initially treated with high-dose prednisone (60 mg/day) and methotrexate. Medication was intensified in case of a DAS >1.6 and tapered in case of a DAS <1.6. The other 124 RA-patients were treated according to routine care, consisting of initial methotrexate and time to IA (n=95). Patient-level Cox regression proportional hazard modelling of associations between maximum observed score per patient for baseline MRI abnormalities and time to IA (n=95) was performed. The hazard ratio (HR) and 95% confidence interval (95% CI) were calculated.

Conclusions: A late flare occurred in 20% of patients receiving intensive treatment but also in more late flares. Together these data do not provide evidence to prioritise the studied intensive treatment strategy above current routine care.

Disclosure of Interest: None declared