Results: Among 96 RA patients, the mean of serum IgG4 was 48.0 ± 45.4 mg/dL and 6.3% had elevated serum IgG4. The mean serum IgG4/IgG ratio of RA patients was $3.5\% \pm 2.8\%$ (range 0.2%–16.9%). There was no patient with elevated serum IgG4 in ankylosing spondylitis, systemic lupus erythematosus, Sjogren's syndrome, and inflammatory myositis. When the patients were divided according to clinical activity, the percentages of the positive serum IgG4 were 25% in active disease group and 4% in low activity group. However, the serum IgG4 levels of the RA patients with active disease activity were not significantly higher than those of the RA patients with low disease activity (58.3 ± 44.3 mg/dL vs. 39.9 ± 30.1 mg/dL). No significant relationship was observed between the ratio of IgG4/total IgG and disease activity. The IgG4 concentrations and total IgG/IgG4 ratios were similar between RA and the other autoimmune diseases ($p > 0.05$).

Conclusions: Our results showed that elevated serum IgG4 in RA is relatively common. However the presence of the elevated serum IgG4 was not associated with disease activity of RA. Further investigations are needed to explore the clinical significance in a larger study population.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.4599

AB0212 DIAGNOSTIC DELAY FOR RHEUMATOID ARTHRITIS: A SYSTEMATIC REVIEW

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Background: Rheumatoid arthritis (RA) is a common inflammatory condition, affecting 1% of the population and causing pain, stiffness and swelling, leading to significant disability and loss of function.¹ Delays in the diagnosis and treatment of RA can lead to worsened joint damage and disability, in addition to a reduced rate of disease-modifying antirheumatic drugs (DMARD)-free remission. Current (2016) EULAR guidelines specify that combination DMARD treatment be initiated within 3 months of the onset of persistent RA symptoms.² Unfortunately, this target is not always achieved due to delays between symptom onset to treatment initiation.

Objectives: The aim of this systematic review, was to determine the extent of delay that occurs at different points in the patient's journey from RA symptom onset to treatment initiation, providing benchmarks of delay.

Methods: Embase and Medline were searched for articles examining diagnostic and treatment delay of RA. To be included, articles had to report a time-period of delay in an adult RA population. Papers were screened by three authors (CAH, JAP, IS). The primary outcome was the reported time-period of delay at any point from RA symptom onset to treatment. Due to skewed delay data, medians (with Interquartile range (IQR)) were selected and reported using narrative synthesis. Different time-periods of delay were categorised to facilitate comparison.

Results: Of 4925 returned articles, 1501 duplicates were removed. The remaining articles were then screened by title, abstract and full text, leaving 26 from which we extracted data. Delay periods were categorised as 1) symptom onset to initiation of DMARDs (n=9), 2) symptom onset to diagnosis (n=14), 3) symptom onset to 1st healthcare professional (HCP) appointment (n=15), 4) 1st HCP appointment to rheumatology referral (n=4) and 5) 1st HCP appointment to diagnosis (n=4). Time-periods of delay were typically skewed to the right. The total delay from symptom onset to receiving DMARDs has dropped since the 1980's (429 weeks before 1987) and by 2014 data indicates an average delay of 23 (IQR 14, 43) weeks. Within this total delay period, delay from symptom onset to diagnosis is at a minimum 16^(7,55) weeks and delay from symptom onset to first contact with a HCP predominantly ranges from 2 (1,8) to 10^(4,24) weeks in data from 2010 onwards. Delay between 1st HCP appointment and Rheumatology referral can be as quick as 2 (1,5) weeks and is within 12^(2,48) weeks across all data points. Delay acquired between 1st HCP appointment and receiving a diagnosis has decreased overtime, most recently, delay was reported as 21 weeks.

Conclusions: Time from RA symptom onset to receiving treatment has reduced considerably in recent decades. However, despite current guidelines and research indicating an optimal treatment window for RA of twelve weeks from symptom onset, this remains unmet, with this delay approximately twice the recommended period. Continued effort is required in reducing delay across all areas of the RA patients' journey to the early treatment needed to improve outcome.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.5439

AB0213 CORRELATION BETWEEN COMPONENTS OF THE DAS28 SCORE AND HEALTH ASSESSMENT QUESTIONNAIRE IN EARLY RHEUMATOID ARTHRITIS

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Background: The health assessment questionnaire (HAQ) in rheumatoid arthritis (RA) has been widely validated as a patient reported outcome measure (PROM). In the United Kingdom regulatory bodies such as the national institute of health-care and clinical excellence (NICE) have been using improvement in HAQ as a surrogate for efficacy of drugs in RA and has informed their calculation of quality adjusted life year, and calculating health costs using incremental costs effectiveness ratios. Most clinical trials in RA report their primary outcome as improvement in clinical parameters such as swollen and tender joints, inflammatory markers and patient and physician global assessment of disease. What is not clear how the two sets of parameters interact. Some reports¹ indicate that there is a strong correlation in early disease, but this has not been validated.

Objectives: We set out to determine the relationship between HAQ scores and clinical parameters of the Disease activity score (DAS28) in addition to physician global.

Methods: Patients were recruited from a single centre from the RAMS study in the North west of England. This is a study of patients with early newly diagnosed RA commencing methotrexate A subset of patients filled in the HAQ questionnaire and this as used as an outcome variable using linear regression and the swollen joints, tender joints, patient global assessment of disease as well as physician assessment of disease in addition to inflammatory markers were used as explanatory variables. These were then adjusted for age.

Results: 81 patients were included in the analysis. median age was 63.1 years (IQR 52.9,72.5). 50 (61.7%) were female. the median HAQ score at baseline was 1 (IQR 0.5,1.5) the median DAS28 score 5.3 (IQR 4.5,6.3). Tender joints at baseline correlated well with HAQ score Beta=0.058 95% CI, 0.04,0.08 ($p < 0.01$). Swollen joints did not correlate with the HAQ beta=0.000 (95%CI -0.3,0.3). Physician global correlated well with disease beta=0.014 (95%CI 0.005,0.022). Patient global assessment also correlated well with HAQ (beta 0.014 95% CI 0.008,0.020). CRP did not correlate with HAQ (beta 0.00261 95% CI -0.002,0.007)

Conclusions: In this small study, patient and physician related outcome measures correlate with HAQ scores at baseline more than measures of joint swelling and inflammatory markers. This indicates that using HAQ as an outcome measure underestimates the effect of treatment. When assessing the efficacy of drugs using HAQ this should be taken into account. Validity of the approach needs to be reviewed.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.5681

AB0214 THE TRAJECTORY OF DISEASE ACTIVITY IN RHEUMATOID ARTHRITIS IN THE FIRST TWO YEARS OF TREATMENT IN AN ASIAN RA COHORT

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Background: Response to disease-modifying antirheumatic drugs (DMARDs) is heterogeneous. Clinical information and baseline characteristics do not allow reliable prediction of which trajectory patients will follow after DMARD initiation.

Objectives: We analysed the change in disease activity over the first two years of treatment in rheumatoid arthritis (RA) to identify different treatment response patterns among RA patients initiating DMARDs. We wanted to establish a predictive model for identifying patients with different treatment response patterns.