fulfilled SLE criteria. The immunological tests showed anti-nuclear antibodies among all patients with high titre and anti-DNA antibodies in SLE and CAH patients respectively. One RA patient had anti-Ro60 antibodies.

Conclusions: During our study, AAA were found fortuitously in AIRD. This association is rarely described in the literature. Hepatic involvement can be seen during AIRD. Nevertheless, a CAH and CD associated shouldn't be disregarded.

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SAT0109 QUANTITATIVE ESTIMATES OF DAMAGE AND DISTRESS, IN ADDITION TO INFLAMMATION, AND THE PROPORTION EACH OF THE 3 VARIABLES AFFECTS CLINICAL MANAGEMENT DECISIONS (TOTAL=100%) MAY CLARIFY ASSESSMENT OF CLINICAL STATUS IN PATIENTS WITH RHEUMATOID ARTHRITIS (RA)

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Background: Quantitative assessment in rheumatoid arthritis (RA) is directed to inflammatory activity (INF) and not to joint damage (DAM) and distress (STR - seen as fibromyalgia, depression, etc.). However, DAM and STR may affect clinical management and outcomes of treatment in many RA patients. For example, an RA patient with well-controlled INF who has secondary fibromyalgia may have 0 swollen joints (SJC) and an ESR≥ of 15, but nonetheless have a DAS28 of 5.1, CDAI of ≥22, and RAPID3 of ≥16 (indicating high activity), based on 14/28 tender joints and a patient global assessment of 80/100. Therefore, quantitative estimates of DAM, and STR, as well as INF may clarify patient status and clinical management decisions.

Objectives: To analyze physician quantitative estimates for the proportion of management decisions attributed to INF, DAM, or STR (total=100%) in RA patients seen in routine care.

Methods: At one academic rheumatology center, the rheumatologist completes four 0–10 visual analog scales (VAS) for overall global assessment (DOCGL), INF, DAM, and STR. In patients with DOCGL ≥2, the proportion of management decisions are estimated as %INF+%DAM+%STR=100%. Cross-tabulations were computed for various phenotypes in 5 INF+DAM and INF+STR categories, 0, 1–20%, 21–40%, 42–60%, and 61–100%.

Results: Among the 77 RA patients, >40% of clinical management decisions were attributed to INF in only 31 (40%), versus >40% to DAM in 33 (43%), and >40% to STR in 17 (22%) (Table). No category of INF+DAM or INF+STR included more than 20% of the patients, and patients were found in 17 of 25 possible categories for combinations of INF+DAM and INF+STR. The 13 patients (17%) in whom INF was estimated to contribute 0% to management included 3 of 5 DAM and 5 of 5 STR categories (Table). The 23 patients with 1–20% of management attributed to INF included 4/5 DAM and 5/5 STR categories. The 10 with 21–40% INF included 4/5 DAM and 4 STR categories. The 16 with 41–60% attributed to INF included 3 DAM and 2 STR categories. Only 15 of the 77 patients (19%) had >60% attributed to INF.

Number among 77 RA patients with physician estimates of % inflammation, % damage and % distress (total=100%) in clinical management decisions (% is of all patients)

% Inflammation:	0	1–20%	21–40%	41–60%	61–100%	Total
N	13 (17%)	23 (30%)	10 (13%)	16 (21%)	15 (19%)	77
% Damage:						
0–20%	3 (4%)	8 (10%)	5 (6%)	3 (4%)	14 (18%)	33 (43%)
21–40%	0	0	2 (3%)	8 (10%)	1 (1%)	11 (14%)
41–60%	3 (4%)	4 (5%)	1 (1%)	5 (6%)	0	13 (17%)
61–80%	0	6 (8%)	2 (3%)	0	0	8 (10%)
81–100%	7 (9%)	5 (6%)	0	0	0	12 (16%)
% Distress:						
0–20%	7 (9%)	13 (17%)	3 (4%)	15 (19%)	15 (19%)	53 (69%)
21–40%	1 (1%)	2 (3%)	3 (4%)	1 (1%)	0	7 (9%)
41–60%	2 (3%)	2 (3%)	2 (3%)	0	0	6 (8%)
61–80%	1 (1%)	4 (5%)	2 (3%)	0	0	7 (9%)
81–100%	2 (3%)	2 (3%)	0	0	0	4 (5%)

Conclusions: Quantitative physician estimates of the proportion of clinical management decisions attributed INF, DAM, and STR may help clarify RA patient status and document a basis for clinical decisions. High levels of DAM and/or STR may explain in part why a target of RA remission often is not met in many patients seen in routine clinical care.[1]

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SAT0110 THE IMPACT OF PUMMONARY INVOLVEMENT TO THE TREATMENT OF REUMATOID ARTHRITIS

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Background: Standard treatment of patients with rheumatoid arthritis (RA) complicated with pulmonary involvements has not been clarified.

Objectives: To evaluate the influence of pulmonary involvement on immunological background and treatment of patients with RA.

Methods: A cross-sectional study was conducted, in which medical records of 479 RA patients who visited our hospital during September the first to November 31th, 2016 were reviewed. Pulmonary involvements were diagnosed by imaging including plain chest radiography or chest computed tomography findings. Patients were divided into two groups, with or without pulmonary involvement, and compared with immunological background (serum level of rheumatoid factor (RF), anti-CCP antibody and recent treatment.

Results: Among 479 patients, pulmonary involvements were diagnosed in 158 patients (female =116), mean age was 73.4 (standard deviation (SD) 9.0) year old, and mean disease duration was 143.3 (SD 125.9) months. Pulmonary involvements included interstitial pneumonia (N=52), organizing pneumonia (N=11), airway diseases (N=36), old tuberculosis (N=18), nontuberculous mycobacteria (N=13), pleurisy (N=5). Higher anti-CCP titers were found in RA with pulmonary involvement than RA only (medians 303.9±596.5 versus 163.5±256.1 U/mL, P<0.001), and the same result was found in RF (medians 322.5±437.2 versus 157.9±277.5 IU/mL, P<0.001). Methotrexate (MTX) was less frequently used (N=56, 35.4% versus N=205, 63.9%, P<0.001), but biological agents were more used (N=31, 19.6% versus N=49, 15.3%), especially abatacept (ABT) was highly used (N=15, 9.5% versus N=8, 2.5%).

Conclusions: RA patients with pulmonary involvements had high immunological response and were less prescribed MTX with may injure lungs and more used ABT. Association between pulmonary involvement and high titer of ACPA, and between positivity for RF or ACPA and good response of ABT were reported. ABT may be useful treatment for RA patients with pulmonary involvement.

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SAT0111 CLARA CELL PROTEIN CC16 AND ITS PATHOGENIC ROLE IN BRONCHIAL OBSTRUCTION IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Bronchial obstruction (BO) is a common manifestation of lung involvement in rheumatoid arthritis (RA) with high incidence from 60 to 80% of all cases. However the pathogenesis of BO in patients with RA remains unknown. Serum level of protein CC16 produced by Clara cells in terminal bronchioles has been reported to decrease in BO associated with bronchial asthma, chronic obstructive pulmonary disease and others. CC16 was considered to demonstrate anti-inflammatory effect via inhibition of interferon-gamma, tumor necrosis factor alpha, interleukin 1 beta, neutrophil elastase and other proinflammatory factors. Also it was shown that CC16 deficiency has a pathogenic effect in BO. In the same time the role of protein CC16 in the pathogenesis of autoimmune diseases (and RA) is not studied.

Objectives: We aimed to evaluate serum level of CC16 in patients with RA in dependence on the presence and severity of BO.

Methods: Serum levels of CC16 in 66 patients with RA and 13 healthy controls were measured by enzyme linked immunoadsorbent assay (ELISA). Patients with RA underwent survey, physical examination and pulmonary function tests (PFTs) including spirometry and bronchodilator test with inhalation of salbutamol (N=41) and body plethysmography (N=11). Statistical processing was carried out using Spearman correlation coefficient and Mann-Whitney test. P value <0.05 was considered as significant.

Results: More than 60% of participants with RA had BO in terminal bronchioles (small airway obstruction), which was revealed with changes of expiratory flows (forced expiratory volume in 1s (FEV1), forced expiratory flow (FEF) between 50% and 75% of forced vital capacity), residual volume (RV) and bronchial resistance (SGaw) in relation to proper values. Depression of post-bronchodilator $FEF_{75\%}$ lower than 70% was adopted as the main criterion of BO. There were no differences (p value >0,05) between serum levels of CC16 in patients with RA (20,14±1,49 ng/ml) and control group (22,70±2,13 ng/ml). However in patient group those with BO had significantly lower levels of CC16 (15,59±1,89 compared with 27,43±2,81 in patients without BO, p value <0,01). Lower CC16 was associated with decreased post-bronchodilator FEV1 and $FEF_{75\%}$ (r =0,345, p