disease, myocardial ischemia, pulmonary hypertension) was evaluated by 2D transthoracic echocardiography (TTE).

Statistical analysis was done in SPSS, $p < 0.05$.

Results Valvular disease was reported in 61.98% SLE (14% stenosis, 20.66% valvular masses, 33% mild-to-moderate regurgitation, 34.73% leaflet thickening. Libman-Sacks endocarditis was not depicted. Asymptomatic decrease in left ventricular ejection fraction (55%) was described in 16.52%, the lowest LVEF being 33% in 4.13% patients; statistical significant negative correlation LVEF – disease duration and activity ($p < 0.05$) was found. LV diastolic dysfunction as subclinical cardiac involvement was registered in 59.50% SLE, with a direct correlation with disease duration ($p < 0.05$), but not with disease activity (SLEDAI) and organ damage (SLIC/ACR) ($p > 0.05$). Global hypokinesia on TTE as an indicator of subclinical myocarditis was demonstrated in up to one third SLE, while cardiomyopathy in one fourth. Mild pericardial effusion is observed in 31.2% cases, while cardiac tamponade in 4 cases; pericardial thickening was reported in 38% SLE. Finally, abnormal systolic pressure in pulmonary artery was found in 24.79% patients.

Conclusion Patients with SLE are at increased risk to develop either clinical or subclinical cardiovascular manifestations as demonstrated by echocardiographic studies. A systematic TTE assessment is routinely recommended for the screening and monitoring of cardiac events.

PS1:19

BIOLOGICAL DMARDS-INDUCED LUPUS IN PATIENTS WITH RHEUMATOID ARTHRITIS: A SINGLE CENTRE EXPERIENCE

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Biological drugs (bDMARDs) have changed landscape and outcomes in various rheumatic conditions. Increased risk for infections, paradoxical reactions, autoimmunogenicity are widely acknowledged and may be responsible for treatment failure and switching among biologics.

Objective To assess drug-induced autoimmunity (serology, clinical significance) in patients with rheumatoid arthritis (RA) treated with bDMARDs.

Patients and methods Longitudinal observational study in 246 bionative RA receiving their first bDMARD, anti-tumour necrosis factor (TNF) or non-TNF drugs, according to local treatment guidelines.

Disease activity, therapeutic response and serial autoantibody profiles (antinuclear (ANA), anti-dsDNA) were systematically assessed at baseline and every six months. Prevalence, clinical significance and therapeutic implications of ANA/anti-dsDNA specificities were further analysed (SPSS-19, $p < 0.05$).

Results 214 RA received TNF inhibitors (56 infliximab, 65 adalimumab, 8 golimumab, 18 certolizumab, 67 etanercept) as first bDMARDs and 32 different mechanism of action (10 rituximab, 7 abatacept, 15 tocilizumab). Overall, ANA seroconversion was demonstrated in 14.22% (35/246), while anti-dsDNA in 8.94% (22/246); no change in the immunological profile under golimumab, certolizumab, abatacept or rituximab.

Significantly higher rates of ANA were reported in infliximab-treated RA (30.53%) as compared to adalimumab (18.46%), etanercept (7.46%) and tocilizumab (6.66%) ($p < 0.05$); anti-dsDNA positivity was found in 10 infliximab (17.85%), 4 adalimumab (12.30%), 3 etanercept (4.47%) and one tocilizumab (6.66%), within an average of 36.5 (2–67) months.

ANA/anti-dsDNA positive status correlated with RA activity (DAS28-ESR) and loss of therapeutic response, concomitant DMARDs (leflunomide) and corticosteroids, administration adverse reactions ($p < 0.05$).

Only four patients (1.62%; 4/246) were classified as drug-induced lupus. Three of them (infliximab, adalimumab, tocilizumab –induced) developed mild to moderate disease, with characteristic malar rash, serositis, haematological, elevated ANA and anti-dsDNA that normalised six months after bDMARD discontinuation; the fourth patient had adalimumab-induced lupus two months after drug initiation, with severe course (subacute extensive skin lesions, cytopenia, serositis) and fatal outcome.

Conclusions Development of ANA with or without anti-dsDNA specificity is typically associated with loss of response to bDMARD in RA and infrequently responsible for drug-induced lupus. Systematic evaluation of ANA/dsDNA is not recommended in routine practice, but essential in certain specific clinical settings (secondary non-responders or lupus-like symptoms).

PS1:20

MINING FOR COMMON REACTIVITY PATTERNS OF HUMAN AUTOANTIBODIES AGAINST ENDOGENOUS PROTEIN TARGETS USING CLUSTERED AUTOANTIBODY REACTIVITIES

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Purpose Autoimmune diseases arise from an abnormal immune response of the body against self-proteins leading to tissue and organ damage. The excessive production of harmful autoantibodies (AAB) is a hallmark of autoimmune diseases including rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), systemic sclerosis (SSc) and Sjogren's syndrome (SjS). Also in cancer research, it has been recently shown that AABs are useful to characterise patients. The characterisation of patient subgroups by means of stratification is essential for the efficient development of therapies, but often difficult due to the lack of appropriate biomarkers. Personalised or precision medicine approaches rely on appropriate multivariate multiplexing technology and data analysis. AABs serve as diagnostic markers for various autoimmune diseases, but the co-occurrence of AABs has rarely been analysed and is difficult to comprehend. Detecting a broad set of AABs helps to investigate the similarity between patients. A multiplex signature enables clustering for the investigation of relationships and patterns, which can be related to relevant clinical variables.

Methods Here, we illustrate Luminex bead-based AAB assays using a set of 96 biomarker targets and their utility to characterise SLE and SSc as well as cancer study groups.

Data analysis is based on bi-clustering algorithms and a prevalence and signature analysis of the markers. Cluster analysis was also performed using transformed data sets (qualitative) to