BIOLOGICAL DMARDS-INDUCED LUPUS IN PATIENTS MINING FOR COMMON REACTIVITY PATTERNS OF LUPUS mab, certolizumab, abatacept or rituximab. (22/246); no change in the immunological profile under golimumb demonstrated in 14.22% (35/246), while anti-dsDNA in 8.94%

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 myocardiitis was demonstrated in up to one third SLE, while cardiomypathy 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.

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

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 bionaive 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 bDMADs and 32 different mechanism of action (10 rituximab, 7 abatacept, 15 tocilizumab). Overall, ANA seroconversion was 63% (67) months. 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).