generic questionnaires (HAQ-DI and SF-36) and a disease-specific measurement RAQoL.
Methods: A total of 220 patients with a mean age 55.05±10.63SD meeting the 1987 ACR classification criteria for RA were included in the study. The mean disease duration was 9.97±5.78SD. Patients were stratified according to treatment regimens into 2 age-matched treatment groups: 96 on csDMARDs and 124 on bDMARD therapy. Subjects with significant comorbidity, infectious disease, congestive heart failure (NYHA class III or IV), malignant hypertension, psychiatric illness, a history of lymphoproliferative disease or neoplasia were excluded from the study. All participants completed the HAQ-DI, SF-36v2TM and RAQoL at baseline, at months 6 and 12 thereafter. The scores of the three instruments were calculated via licensed calculator. Comparison was performed by analysis variance ANOVA.
Results: At baseline the mean scores of HAQ-DI and RAQoL did not differ greatly among patients on csDMARDs and bDMARDs (1.29±0.78SD *vs* 1.13±0.54SD, p=0.063; 16.31±8.26SD *vs* 15.03±7.13SD, p=0.219, respectively). However, the mean physical component summary score of SF-36 was significantly higher in bDMARDs compared with csDMARDs (32.98±5.97 *vs* 31.05±7.82, p=0.039), while in the mental component of this scoring system not such a difference was found (p=0.983). After 6 months subjects treated with bDMARD showed a significant decreasing of the means of the HAQ-DI and RAQoL, as opposed to the other treatment group (0.86±0.5SD *vs* 1.17±0.76SD, $p<0.001$; 10.98±6.53SD *vs* 14.55±7.96SD, $p<0.001$ respectively). Similar results were obtained for both physical and mental component summary scores of SF-36 (39.49±6.43SD *vs* 33.48±8.04SD, $p<0.001$; 43.69±7,99SD *vs* 39.66±10.19SD, p=0.001 respectively). At month 12 a significant improvement of QoL, measured by the three assessment tools was registered in patient receiving bDMARDs compared with the csDMARD treatment group ($p<0.001$).
Conclusions: Patient treated with bDMARDs showed better results for QoL than those on therapy with csDMARDs within a period of 12 months of treatment. Current management strategies should focus on improving the symptoms of activity and maintaining physical function in order to increase QoL in patients with RA.
Disclosure of Interest: None declared
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AB0284 ASSESSMENT OF DISEASE ACTIVITY BY DAS28-CRP, CDAI, SDAI AND RESPONSE TO TREATMENT WITH CSDMARDS AND BDMARDS AFTER ONE-YEAR FOLLOW-UP IN RHEUMATOID ARTHRITIS PATIENTS FROM BULGARIAN POPULATION

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Background: The assessment of disease activity is an essential component in the selection of therapeutic approach for the prevention of disability of patients with RA.
Objectives: The current study was conducted to evaluate the disease activity in patients on csDMARDs and bDMARDs after 6 months to 1-year of treatment and to determine whether the benefits of different therapies were sustained over time.
Methods: For the purpose of the study were selected 220 patients with a mean age 55.05±10.63SD years, meeting the 1987 ACR classification criteria for RA. Patients were stratified according to treatment regimens into 2 age-matched treatment groups: 96 on csDMARDs and 124 on bDMARD therapy. Patient's assessment of disease related pain, global health and physician assessment of global health was made by visual analogue scale (VAS) – 100mm. Disease activity was the primary outcome domain. Independent joint assessor evaluate 28 joints on baseline, 6th and 12th month of the follow-up period. C-reactive protein (CRP) was used to measure the inflammation process. DAS28-CRP, CDAI and SDAI were calculated according to the standard formulas. Comparison was performed by analysis variance ANOVA.
Results: On baseline, patients on bDMARDs had a significantly higher mean time-averaged 28-joint disease activity score (5.03±0.84SD vs. 4.35±1.20SD, $p<0,001$), CDAI (25.06±7.32SD vs. 20.83±10.53SD, $p<0.001$) and SDAI (28.27±8.74 SD vs. 23.19±11.89 SD, $p<0.001$) compared to those on csDMARDs. On the 6th month in both groups (bDMARDS and csDMARDs) we found significant decrease in mean DAS28 ($p<0.001$, $p<0.001$), although no significant difference in disease activity between the groups was measured by this indicator (3.75±2.49 SD vs 3.90±1.10 SD, p=0.566). Patients on bDMARDs had significantly lower disease activity compared to those on csDMARDs after 6th and 12th month of treatment assessed by CDAI (13.43±4.98 SD vs 16.81±9.94 SD, p=0.001; 8.65±4.53 SD vs 15.86±10.02 SD, $p<0.001$), and SDAI (14.63±5.42 SD vs 18.38±10.49 SD, $p<0.001$; 9.39±4.92 SD vs 16.79±10.5 SD, $p<0.001$). Unlike results reported by DAS28-CRP which showed no change between the 6th and 12th month in patients receiving csDMARDs (3.90±1.10 SD, 3.82±1.12 SD, p=0.156) we observed a statistically significant difference in all three time intervals (0,6th,12th month) of the follow up period regarding to CDAI and SDAI.
Conclusions: After a year prospective follow-up, therapy with biologic DMARDs results in sustained suppression – minimal disease activity assessed by DAS28-CRP, CDAI and SDAI, compared to patients receiving DMARDs who had moderate disease activity according to these tools. The therapy with bDMARDs was superior to csDMARDs therapy for suppressing disease activity (assessed by DAS28-CRP, CDAI and SDAI) of rheumatoid arthritis (RA) on 6th and 12th month of the follow-up period.
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AB0285 CLINICAL SIGNIFICANCE OF GLUCOCORTICOID-INDUCED TUMOR NECROSIS FACTOR RECEPTOR RELATED PROTEIN LIGAND (GITRL) IN RHEUMATOID ARTHRITIS: RELATION TO DISEASE ACTIVITY AND TREATMENT OUTCOME

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Background: Glucocorticoid-induced tumor necrosis factor receptor related protein (GITR) is a member of the tumor necrosis factor receptor superfamily that is activated by its specific ligand (GITRL). GITR is mainly expressed in immature and mature T cells especially regulatory (Treg) cells (CD4+ CD25+) and effector T cells (CD25–) [1]. GITRL is mainly expressed in endothelial cells, dendritic cells, macrophages and B cells but not in T cells. GITR-GITRL system is known to have important regulatory role on inflammatory response and immune reactivity.
Objectives: This study aimed to measure serum and synovial fluid (SF) levels of GITRL in patients with recent onset rheumatoid arthritis (RA) before and after initiation of therapy and to evaluate the relationship between GITRL and RA clinical and laboratory characteristics, disease activity and response to therapy.
Methods: We measured GITRL in the serum (n=48) and SF samples (n=21) from 48 recent onset RA patients and in the serum from 20 healthy control (n=20) at baseline and 6 months after initiation therapy with non-biological disease modifying anti-rheumatic drugs (DMARDS). In the patients Disease activity was calculated by the 28 joint counts (DAS28) and musculoskeletal ultrasound examination (MSUS) was performed at baseline and after 6 months using a 12-joint score (bilateral elbow, wrist, 2nd metacarpophalangeal (MCP), 3rd MCP, knee, ankle) [2]; immunoglobulin-M rheumatoid factor (IgM-RF) titre, anti-cyclic citrullinated peptide (anti-CCP) antibodies titre and C-reactive protein (CRP) levels were measured and the health assessment questionnaire (HAQ) score were recorded.
Results: Serum and SF GITRL levels were highly significantly increased in RA (39.38±16.78 ng/mL and 30.6±16.79 ng/mL respectively) compared to serum level in the healthy controls (10.3±5.46 ng/mL) ($p<0.001$). In RA patients, baseline serum and SF levels of GITRL significantly correlated with DAS28 (r=0.52 and 0.56 respectively, $p<0.05$), anti-CCP titres (r=0.46 and 0.51 respectively, $p<0.05$), grey scale (GS) (r=0.5 and 0.52 respectively, $p<0.05$) and power Doppler (PD) (r=0.65 and 0.68 respectively, $p<0.001$) synovitis scores. Also, serum and SF levels of GITRL at 6 months follow up significantly correlated with the DAS28 (r=0.42 and 0.48 respectively, $p<0.05$), GS score (r=0.46 and 0.51 respectively, $p<0.05$), PD signal (r=0.43 and 0.45 respectively, $p<0.05$). Logistic regression analysis showed that baseline serum levels of GITRL were predictive of follow up DAS 28 and PD synovitis score (p=0.009 and 0.03 respectively).
Conclusions: Rheumatoid arthritis patients have significantly increased serum and synovial levels of GITRL that remarkably correlated with the DAS28 and MSUS parameters of inflammations suggesting that it could be a useful marker to reflect RA disease activity. GITRL could be a useful biomarker to predict treatment outcome in RA patients.
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AB0286 14 CASES STUDY OF MACROPHAGE ACTIVATION SYNDROME (MAS) IN SYSTEMIC ONSET JUVENILE IDIOPATHIC ARTHRITIS

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Background: Macrophage activation syndrome (MAS) is a severe, potentially life-threatening syndrome.
Objectives: we aim to review the precipitating events,clinical features, treatment, and outcome of macrophage activation syndrome (MAS).Part patients were analysed the Polymorphisms of Perforin A91V (NCBI:SNP rs35947132) using special primers by polymerase chain reaction (PCR).
Methods: Retrospective review of cases of MAS from a prospectively collected database of children with autoimmune diseases from 2003 to 2008.
Results: Fourteen patients (nine boys) were considered to have evidence of MAS.The primary diagnosis was systemic onset juvenile idiopathic arthristis, with age ranged from 5 months to 12 years. No medication was identified as trigger. Eleven had infections prior to MAS,specific infectious agents were identified in four. High grade fever, new onset hepatosplenomegaly, lymphadenopathy, dysfunction

of liver,abnormal fat metabolism and hemophagocytosis were common clinical features.Two cases were with ARDS and MOF in three and three died. The perforin A91V (NCBI:SNP rs35947132) gene was detected in seven systemic onset juvenile idiopathic arthritis compolicated with MAS cases, but no mutation were found. Glucocorticoid, intravenous immunoglobulin, immunoimpressive therapy were effective and HP (Plasmapheresis) used in one serious case was also effective.
Conclusions: MAS is a rare and potentially fatal complication of childhood rheumatoid diseases,especially systemic onset juvenile idiopathic arthritis. Most of our patients were male, and most cases were preceded by infection. Bone marrow studies support the diagnosis. MOF may be a poor prognostic sign.Aggressive early therapy is essential.
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AB0287 MUTUAL ASSENT TOWARDS COMPREHENSIVE DISEASE CONTROL: THE RELATIONSHIP BETWEEN US MEASURES AND PATIENT REPORTED OUTCOMES IN EARLY RHEUMATOID ARTHRITIS

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Objectives: Assessment of the relationship between US measures of joint inflammation/damage and patient reported outcomes (PROs): HAQ, pain and patient global assessment in early rheumatoid arthritis (early RA) patients over 5-years follow up period.
Methods: This longitudinal cohort of 261 patients with early RA was derived from the US monitoring study [1]. Adopting OMERACT definitions; correlations between total US scores (synovial hypertrophy, synovial fluid, Power Doppler, bone erosion and tenosynovitis) and PROs [2] namely functional disability (HAQ), pain and patient global scores were determined at 0, 1, and 5years. Radiological damage was assessed using modified Total Sharp score (mTSS). Univariate correlations as well as correlations between interval changes were assessed. Multivariable regression models were used to evaluate the associations over all time-points and their relationship to clinical disease activity measures.
Results: There were significant correlations ($p<0.01$) between total US score and HAQ (r=0.71), pain (r=0.69) and patient global scores (r=0.66) at all timepoints. The association tends to be stronger with increase disease duration (Spearman correlation 0.12 at baseline, 0.22 at 1-year and 0.41 at 5-years). Change in mTSS score at 5-years was not associated with changes in PROs. Improvements in US scores were also associated with improvements in PROs. Multivariate models revealed that synovial hypertrophy and Power Doppler scores were associated ($p<0.01$) with functional disability, pain and patient global assessment, controlling for clinical disease activity measures. Studying the pattern of joint involvement, it was associated significantly ($p<0.01$) with the US score of the affected joints. US total score at 1-year predicted subsequent 5-year HAQ score (R2=0.17). At 0, 1- and 5-years, total US scores were higher in patients whose HAQ score was >1 (9.26) compared to those below 1 (4.16, $p<0.01$).
Conclusions: the link between joint inflammation/structural damage and PROs is of critical importance to the care of patients with inflammatory arthritis. US measures of inflammation and structural damage correlated independently with physical function, pain and patient global assessments. A clear relationship between radiographic structure damage and the patient's perceived remission/flare provide the basis for comprehensive disease assessment and management.
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AB0288 LABORATORY MARKERS OF INFLAMMATION AND SERUM NICOTINAMIDE PHOSPHORIBOSYLTRANSFERASE LEVEL IN RHEUMATOID ARTHRITIS PATIENTS

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Objectives: To study relationship between serum levels of nicotinamide phosphoribosyltransferase and laboratory markers of inflammation in patients with rheumatoid arthritis (RA).
Methods: We determined nicotinamide phosphoribosyltransferase level in sera of 140 patients with RA (96 women and 44 men) by indirect enzyme-linked immunosorbent assay (RaiBiotech, cat No. EIA-VIS-1). The control group consisted of 20 women and 10 men aged 22 to 55 years without complaints of pain in the joints throughout life. The mean duration of disease was 5.94±0.37 years.
Results: We divided all RA patients into 2 groups: one group (118 patients) with elevated levels of nicotinamide phosphoribosyltransferase serum (more than 3.9 ng/ml) and second group (22 patients) - with normal range.
In each of the two groups, the levels of CRP and ESR were determined.
Patients with elevated levels of Nampt had the following laboratory parameters (M±m): ESR – 37.83±1,57, CRP – 56.09±3,73 (rate - less than 5.0 mg/l). The second group had following data: ESR 22.46±0,56, CRP 21,65±1.38. Thus, patients with elevated levels of nicotinamide phosphoribosyltransferase had significantly higher concentrations of ESR and CRP ($p<0,001$).
Conclusions: There is the relationship between the level of nicotinamide phosphoribosyltransferase serum and laboratory markers of inflammation in RA (CRP and ESR). The data indirectly confirm the hypothesis that increased levels of nicotinamide phosphoribosyltransferase in RA patients is associated with disease activity.
Disclosure of Interest: None declared
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AB0289 PREDICTOR OF THE SIMPLIFIED DISEASE ACTIVITY INDEX 50 (SDAI 50) AT MONTH 6 DURING BDMARDS TREATMENT IN PATIENTS WITH LONG-ESTABLISHED RHEUMATOID ARTHRITIS: A SINGLE-CENTER, RETROSPECTIVE STUDY

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Background: The simplified disease activity index (SDAI) 50 has good agreement with the EULAR response measures for early rheumatoid arthritis (RA). Although there are reports on early RA, there have been no reports on long-established RA.
Objectives: In this study, we analyzed the relationships between various baseline factors and SDAI 50 after six months of biological disease-modifying antirheumatic drugs (bDMARDs) treatment to determine the prognostic factors for long-established RA.
Methods: The subjects were 332 RA patients who had been treated with bDMARDs for 6 months. The following characteristics were investigated: age, gender, disease duration, smoking history, body mass index, steroid and methotrexate dosage, previous bDMARDs use, combined csDMARDs use, ESR, CRP, serum matrix metalloproteinase-3 levels, SDAI score, health assessment questionnaire disability index score (for daily living activities) and short form-36 score (for quality of life). As a primary outcome index, SDAI response was defined as a 50% reduction in SDAI score between baseline and 6 months (SDAI 50).
Results: The group of RA patients who achieved SDAI 50 (Group A: 204 patients) had a higher tender joint count (p=0.041), swollen joint count (p=0.001), evaluator's global assessment (p=0.027) and SDAI (p=0.006) than did those who did not achieve SDAI 50 (Group B: 152 patients). Before the start of the treatment, steroid dosage (p=0.0187, odds ratio: 1.119, 95% CI: 1.029–1.229) and SDAI (p=0.0003, odds ratio: 0.953, 95% CI: 0.928–0.978) were determined based on logistic regression analysis. Comparisons were performed between Groups A and B and between before treatment and after 6 months of SDAI. Group A showed a significant improvement compared to Group B by repeated measure analysis of variance (ANOVA) (Interaction: p=0.000, Group A vs. Group B: p=0.000, before vs. after treatment: p=0.000).
Conclusions: Our study demonstrated that RA patients with a lower steroid dosage and higher SDAI baseline are more likely to achieve SDAI 50 with bDMARD treatment.
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Rheumatoid arthritis - comorbidity and clinical aspects

AB0290 PULMONARY AMYLOIDOSIS IN RHEUMATOID ARTHRITIS – A POSTMORTEM CLINICOPATHOLOGIC STUDY OF 161 PATIENTS

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Background: AA amyloidosis (AAa) is one of the most insidious systemic complications of rheumatoid arthritis (RA), which furtively may lead to death [1].
Objectives: The aim of this study was to determine the prevalence and location of amyloid A deposition in the lungs of RA patients at the time of death.
Methods: A randomized autopsy population of 161 in-patients with RA was studied. AAa complicated RA in 34 (21.1%) cases [1].