Background

Objectives To investigate the effectiveness of electrical stimulation on quadriceps and hamstring muscle performance in patients with patellofemoral pain.

Methods Twenty-one patients participated in the 4-week exercise program and were randomly assigned to either closed kinetic chain exercises or closed kinetic chain exercises combined with electrical stimulation of bilateral quadriceps and hamstring muscles. Quadriceps and hamstring muscle peak torques were measured at baseline and at 4 weeks with an isokinetic dynamometer at 60°/sec and 180°/sec angular velocities.

Results After the 4-week exercise program the mean torque values for quadriceps and hamstring at both angular velocities were higher in both groups. There was a significant difference in the left hamstring peak torque values at 60°/sec and 180°/sec angular velocities and in the left quadriceps peak torque at 180°/sec angular velocity in the electrical stimulation group ($p < 0.01$). In the other group the peak torque differences in the left hamstring and quadriceps muscles at 60°/sec angular velocity and in the left hamstring muscle at 180°/sec angular velocity were statistically significant ($p < 0.05$).

Conclusion Although it seems that electrical stimulation does not provide an additional effect on muscle performance, it can be used with closed kinetic chain exercises to accelerate the rehabilitation program.

Humoral aspects of autoimmune disease

AB0016 WEAK CORRELATION OF CIRCULATING IMMUNE COMPLEXES IN SERA OF PATIENTS WITH CONNECTIVE TISSUES DISEASE MEASURED WITH POLYETHYLENE GLICOL AND C1Q BINDING ASSAY

JM Nedovic, SK Stojanovic, BN Stamenkovic, AM Stankovic, AN Dimic. *Rheumatology, Institute for Prevention, Treatment and Rehabilitation of Rheumatic and Cardiovascular Diseases ? Niska Banja, Medical Faculty, University of Nis, Nis, Yougoslavia*

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Background Circulating immune complex (CIC) are forming in a normal immune response, as a consequence of attachment of specifically reactive antibodies to antigens. As a result, most antigens are neutralised and/or eliminated. However, in some circumstances, CIC deposits in tissues with subsequent phlogogenic responses.

Systemic lupus erythematosus (SLE), systemic sclerosis (SSc) and rheumatoid arthritis (RA) are organ nonspecific autoimmune diseases with a high prevalence of positive test for CIC. It seems that systemic character of these diseases is due to vascular entrapment of CIC in various tissues.

Application of various methods on the same serum sample regularly gives a different level of CIC.

Objectives Our objective was to correlate the levels of CIC measured with two different assays in sera of patients with SLE, SSc, and RA.

Methods We used precipitation test with polyethylene glycol (PEG), molecular weight 6000 and C1q binding assay (C1q) with laser nephelometer in sera of 74 patients with SLE, 40 patients with SSc and 42 patients with RA.

Results PEG revealed abnormally high levels of CIC in 62,16% SLE sera, 45% SSc sera and 47,61% RA sera. C1q was more sensitive in SLE (78,37%) and RA (76,1%) with exactly the same result in SSc (45%).

The correlation of these two methods was weak, $r = 0,089$ for SLE, $r = 0,22$ for SSc, and $r = 0,29$ for RA.

Conclusion These results are not only the consequence of the imperfection of these methods but also the consequence of the enormous heterogeneity of CIC.

C1q binding assay is more sensitive, is WHO standardised, but partially gives false positive results in presence of DNA, CRP and RF, which are frequently present in sera of patients with connective tissues diseases. Besides, C1q does not detect CIC with isotypes of immunoglobulins that does not activate complement.

Precipitation test with PEG detects immunoglobulins of all isotypes, does not depend from complement and RF, and is simple and inexpensive to perform. Besides, PEG is not standardised, is less sensitive and partially gives false positive results in state of hypergammaglobulinemia, also frequently present in patients with connective tissues diseases.

This holds the allegations of WHO Working Group for the necessity of using more methods for detecting CIC from the single sample sera.

AB0017 ANTINEUTROPHIL CYTOPLASMIC ANTIBODIES IN RHEUMATOID ARTHRITIS PATIENTS

¹B Sonel, ¹N Suldur, ¹P Ataman, ²H Tutkak, ²N Duzgun, ¹MB Atay. *¹Physical Medicine and Rehabilitation; ²Clinical Immunology and Rheumatology, Ankara University, School of Medicine, Ankara, Turkey*

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Background Antineutrophil cytoplasmic antibodies (ANCA) have been suggested to occur in rheumatoid arthritis (RA), especially in patients with severe and longstanding disease. However in some studies no association was found between disease activity and ANCAs.

Objectives To determine the occurrence of ANCAs in patients with RA and compare the clinical and laboratory activity parameters between ANCAs positivity and negativity.

Methods Sera from 85 patients (mean age 50.5 years, 67 women and 18 men) with RA were included in study. Morning stiffness duration, pain intensity (Visual Analogue Scale), fatigue, tender and swollen joint counts (28 joints), systemic symptoms, patients and physicians general health assessment (Visual Analogue Scale), disease activity (DAS28), functional status (ACR) and disability (Health Assessment Questionnaire) were assessed of all patients.

Besides the routine laboratory activity parameters, ANCA testing was performed by standard indirect immunofluorescence. Antibody against the cytoplasmic antigens proteinase-3 (PR3) and myeloperoxidase (MPO) were measured by enzyme-linked immunosorbent assay.

Results ANCAs were found in 15 (17.6%) of patients with RA; 9 with atypical ANCA, 5 with perinuclear ANCA and 1 with cytoplasmic ANCA. Five of these 15 patients had vasculitis. None of the patients had Feltys syndrome, neutropenia or renal involvement. 4/5 patients with perinuclear ANCA and 1/9 patients with atypical ANCA had positive test result for MPO. None of the patients had PR3.

There were no significant differences in ANCA positivity with respect to the duration of disease (mean 8.7 years), age, gender, clinical and laboratory inflammatory parameters except for the duration of morning stiffness ($p = 0.04$) and antinuclear antibody positivity ($p = 0.001$).

Conclusion We observed that perinuclear ANCA pattern is specific for MPO but perinuclear and atypical ANCAs may not be associated with vasculitis in the presence of RA. Clinical

significance of positive ANCA was not justified with the findings of the study, as the number of patients with positive ANCA was few.

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THU0055 ROLE OF ANTIBODIES TO 5'NUCLEOTIDASE IN RHEUMATOID ARTHRITIS, SYSTEMIC LUPUS ERYTHEMATOSUS, SYSTEMIC SCLEROSIS, ANKYLOSING SPONDYLARTHROSIS AND REACTIVE ARTHRITIS PATIENTS

AB Zborovsky, BV Zavodovsky, EV Bobicheva, LE Sivordova, NA Fofanova. Laboratory for Arthritis and Arthrosis, Research Institute for Clinical and Experimental Rheumatology, Volgograd, Russia

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Background

Objectives Purpose of research was to study presence and role of definition of antibodies to 5'nucleotidase (anti-5NT) in rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), systemic sclerosis (SS), ankylosing spondylarthritis (AS) and reactive arthritis (ReA) patients.

Methods There were 100 RA, 30 SLE, 20 SS, 30 AS and 20 ReA patients and 30 healthy donors (HD) under our observation. Anti-5NT were determined in serum of a blood with ELISA-test. We used 5'nucleotidase (AN 3.1.3.5) from *Crotalus adamanteus* venom («Sigma») as antigen.

Results We revealed, that the anti-5NT level in HD was equal $0,048 \pm 0,004$, in RA – $0,153 \pm 0,012$, in SLE – $0,101 \pm 0,016$, in SS – $0,117 \pm 0,017$, in AS $0,118 \pm 0,015$, in ReA – $0,117 \pm 0,010$ (in all cases $p < 0.05$). We revealed difference between anti-5NT level in RA and SLE ($p < 0,05$), that can be used in differential diagnostics of these diseases. Was noted increasing of anti-5NT level in RA with muscles injury (without myositis – $0,150 \pm 0,013$, with myositis – $0,190 \pm 0,002$) and kidney manifestations (without nephropathy – $0,149 \pm 0,013$, with nephropathy – $0,205 \pm 0,013$). Taking into account that kidney injury in RA associates with perinuclear antineutrophil cytoplasmic antibody, we assume presence of cross reactions between them and anti-5NT.

Conclusion Thus, we revealed the new group of autoantibodies. The determination of them can improve diagnostics and differential diagnostics and better understanding of rheumatic diseases pathogenesis.

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THU0056 EVIDENCE OF B CELL ACTIVATION IN KAWASAKI DISEASE

D Kim. Pediatrics, Yonsei University College of Medicine, Seoul, South Korea

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Background Kawasaki disease (KD) is an acute systemic vasculitis that primarily affects infants and young children below 5 years old. The cause of KD is still unknown, but the acute phase of KD is often accompanied by the alteration of circulating immune cells. The polyclonal activation of B cells during the course of disease is well documented.

Objectives In this study, the evidence of B cell activation and the possible involvement of superantigen in KD were sought.

Methods

Results In 13 out of 15 KD patients, an increase in B cells expressing the VH3 family was seen during the acute phase of disease. Analysis of B cell clonal expansion showed the VH6 family clone of 9 amino acids to be the most common, observed in 5 out of 15 KD patients. Analysis of CDR3 size profile showed that various prominent bands appeared during the acute phase of disease, and some disappearing during the subacute phase, while other newly developed bands appeared during the subacute phase of disease. The DNA sequences of CDR3s showed no dominant clone.

Conclusion These data suggest that KD may be caused by conventional antigen (s).

THU0057 DISTURBED PERIPHERAL B CELL HOMEOSTASIS IN SLE

¹T Dörner, ²M Odendahl, ¹A Jacobi, ¹A Hansen, ¹F Hiepe, ¹GR Burmester, ³PE Lipsky, ²A Radbruch. ¹Department Medicine, Charité, Berlin, Germany; ²Deutsches Rheumaforschungszentrum, Charité, Berlin, Germany; ³NIAMS, NIH, Bethesda, MD, USA

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Background The distribution of peripheral B cell subpopulations in SLE is unknown, although a variety of humoral abnormalities are known.

Objectives The phenotype and Ig heavy chain gene usage of peripheral B cell subpopulations of patients with active systemic lupus erythematosus (SLE) were examined.

Methods Peripheral B cells were characterised by FACS analysis and B cell subpopulations of one patient were characterised for V(D) J gene rearrangements.

Results In patients with SLE, there was a marked B lymphocytopenia that affected CD19⁺/CD27⁻ naïve B cells more than CD19⁺/CD27⁺ memory B cells leading to a relative predominance of CD27 expressing peripheral B cells. In contrast, CD27high/CD38⁺/CD19dim/surface Iglow/CD20-syndecan-1 +plasma cells were found at an enhanced frequency in active but not inactive SLE patients. Upon immunosuppressive therapy, CD27high plasma cells and naïve CD27⁻ B cells were markedly decreased in the peripheral blood. Mutational analysis of V gene rearrangements of individual B cells confirmed that CD27⁺ B cells coexpressing IgD were memory B cells preferentially using VH3 family members with multiple somatic mutations. CD27high plasma cells showed a similar degree of somatic hypermutation, but preferentially employed VH4 family members.

Conclusion These results indicate that there are profound abnormalities in the various B cell compartments in SLE that respond differently to immunosuppressive therapy.