Background YKL-40 or gpl-39 is a surface glycoprotein of connective tissue cells in particular of monocytes, fibroblasts, synovocytes and chondrocytes. According to some Authors, being augmented in serum of rheumatoid arthritis (RA) and, to a lesser extent, of osteoarthritis (OA) it may correlate with the degree and the speed of cartilage degradation.

Objectives To better evaluate if this is true or if this marker is mainly an inflammation index, we have investigated a group of RA patients correlating YKL-40 serum values with some of the clinical parameters of the disease most widely used in literature (swollen or painful joint count, HAQ, PCR, Ra-test, ESR, Rx score of the hands, DAS 28).

Methods We have evaluated 52 consecutive patients (43 F, 9 M) with RA according to the ACR criteria mainly in the 2nd (22) or 3rd (25) functional class of Steinbrocker. Their mean age was 59.7 years (33–86) and the mean disease duration 13.6 years (1–35). The YKL-40 was determined in serum according to the ELISA sandwich method of Harvey *et al*, 1998 (normal range: 25–95 ng/ml); all the patients (pts) were previously visited and all the parameters listed above measured. Moreover the Rx score of the hands was calculate according to the method of Kaye, 1987, slightly modified. The significance of the correlations were evaluated with the Spearman test.

Results The mean ($\pm$ SD) serum values of YKL-40 was 147.84 ($\pm$ 97.47) ng/ml (normal range: 25–95 ng/ml). The correlations with the parameters listed above were those shown in the following Table 1. Swollen joint count, Rx score and Ra-test showed a still less significant correlation with the marker investigated.

Abstract THU0060 Table 1

	r	p
PCR	0.531	< 0.001
DAS 28	0.358	= 0.010
PAINFUL JOINT COUNT	0.322	= 0.020
HAQ	0.260	= 0.056
ESR	0.243	= 0.083

Conclusion Since YKL-40 in RA serum seems to better correlate with PCR, DAS 28 and painful joint count respectively, in our opinion it behaves more like an activity marker of the disease (perhaps of synovitis) rather than of cartilage degradation as claimed by some Authors. Despite this limitation, further studies are needed to evaluate its potential prognostic activity as predictor of joint lesions in RA.

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THU0061 RIBOSOMAL P PROTEIN ANTIBODIES

SM Ivanova, GG Karpova, NV Bogachiova, TA Riazantcva, AI Speransky. *Department of Clinical Immunology, Institute of Rheumatology, Moscow, Russia*

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Background Circulating autoantibodies are one of the main characteristic of autoimmune disease as systemic lupus erythematosus (SLE). The associations between anti-DNA antibodies and renal disease, anti-Ro with cutaneous manifestations and congenital

heart block are well established. The human ribosomal P proteins (RPP) designated PO (38 kd), P1 (19 kd), and P2 (17 kd), the clinical significance of these antibodies is disputed, but some data suggest an association with aggressive disease, nephritis, central nervous system (CNS) abnormalities.

Objectives The aim of the present study was to determine the possible clinical and immunological associations of anti-RPP antibodies in SLE.

Methods Serum samples from 68 patients with SLE who satisfied the American Rheumatism Association revised criteria for SLE entered the study. Anti-RPP activity was determined by ELISA (TRINITY BIOTECH RIBOSOMAL P 1gG, A, M), the specificity was confirmed by Western blotting (Wb). Antinuclear factor (ANF) was detected by indirect immunofluorescence, anti-DNA - by ELISA, anti-Ro, La, RNP, Sm antibodies - by double immunodiffusion, CRP, C3, C4 - by radial immunodiffusion.

Results Anti-RPP antibodies were demonstrated by ELISA in sera of 13 patients (19%). 17 sera were investigated by Wb, 6 of them were positive by both ELISA and Wb, another 11 were negative as in ELISA, as in Wb. Anti-P reactivity was against 38 kd (PO), 19 kd (P1) and 17 kd (P2) in one sera; 38 kd and 17 kd (PO, P 2) in 4 sera; and 38 kd (PO) also in one sera. SLEDAI score was 13,6+ 8,65 in seropositive group, and 16,7+25,82 in seronegative. The frequency of nephritis, CNS involvement was significantly higher in RPP-positive patients-38,5% (5/13) and 38,5% (5/13) against 7,3% (4/55) and 18%(10/55). In the group of 15 SLE patients with CNS involvement 5 (33%) had increased anti-RPP antibody levels. The highest levels of anti-RPP had 3 patients with aggressive disease, all of them had nephritis, one also psychosis-convulsions, and one-hemolytic anaemia.

Conclusion In conclusion, we suggest that anti-RPP antibodies may be a good serological marker for not only SLE, but for aggressive duration of disease, including damaging of kidney and brain.

(abstract for oral presentation).

THU0062 ASSESSING INFLAMMATORY ACTIVITY IN RHEUMATOID ARTHRITIS AND ANKYLOSING SPONDYLITIS. SERUM AMYLOID A OR C-REACTIVE PROTEIN?

¹A Gómez, ¹R Sanmartí, ²L Álvarez, ¹G Salvador, ¹JD Cañete, ²X Filella, ¹J Gratacós, ¹J Muñoz-Gómez. ¹Rheumatology; ²Biochemistry, Hospital Clínic, Barcelona, Spain

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Background Serum amyloid A (SAA) protein has been suggested to be a more sensitive marker of disease activity than CRP in rheumatoid arthritis (RA). Few studies are carried out in patients with ankylosing spondylitis (AS).

Objectives To analyse if SAA correlates with other markers of inflammatory activity such as CRP, ESR and serum levels of IL-6 in both RA and AS. To evaluate if SAA is the most sensitive marker of clinical disease activity in RA.

Methods Using a sensitive ELISA test (N-latex SA, Dade Behring), SAA was measured in the serum of 51 patients with RA and 44 patients with AS. CRP, ESR and serum IL-6 (ELISA) were measured simultaneously. Clinical parameters of disease activity (number of painful and swollen joints) and Thompson's articular index were analysed also in RA patients.

Results The magnitude of the SSA response was greater than the CRP ($x \pm$ SD: SAA = 32.7 ± 56.7 mg/l vs CRP = 18.8 ± 26.3 mg/l $p < 0.001$). Patients with RA showed higher SAA levels than AS ($x \pm$ SD: 44.8 ± 64 mg/l vs 13.6 ± 18.9 mg/l $p <$

0.001). In both RA and AS, SAA correlates significantly with ESR ($r = 0.51$; $p = 0.0001$ in RA and $r = 0.61$; $p = 0.0001$ in AS), CRP ($r = 0.70$; $p = 0.0001$ in RA and $r = 0.66$; $p = 0.0001$ in AS) and IL-6 ($r = 0.58$ in RA; $p = 0.0001$, and $r = 0.48$ in AS; $p = 0.001$). In patients with RA, SAA but not CRP, ESR or IL-6, showed a positive, although weak correlation with the number of painful joints ($r = 0.31$; $p = 0.03$). Thompson articular index, but not the swollen joint count, correlates with the four parameters analysed; this correlation was better for CRP ($r = 0.51$; $p = 0.0001$) than for SAA ($r = 0.39$; $p = 0.007$) ESR ($r = 0.35$; $p = 0.02$) or IL-6 ($r = 0.37$; $p = 0.01$). **Conclusion** In both RA and AS, SAA correlates well with other laboratory markers (mainly with CRP) of inflammation. However SAA is not a better marker of clinical disease activity than CRP in RA.

THU0063 AUTOANTIBODIES TO HUMAN MYC FAR UPSTREAM ELEMENT-BINDING PROTEIN IN PATIENTS WITH RHEUMATOID ARTHRITIS

¹T Nojima, ¹A Suwa, ¹M Hirakata, ¹Y Ikeda, ²T Mimori. ¹Internal Medicine, Keio University School of Medicine, Tokyo, Japan; ²Clinical Immunology, Kyoto University, Kyoto, Japan

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Background We have shown that sera from patients with rheumatoid arthritis (RA) contain a variety of autoantibodies to unknown proteins such as 60-, 45-, and 75-kDa proteins, in preliminary immunoblot studies using HeLa cell extracts.¹ By molecular cloning of the target antigens, we have demonstrated that calpastatin (endogenous protein inhibitor of calpain, calcium-dependent cysteine proteinase) is one of such autoantigens and the presence of anti-calpastatin antibodies may be related to pathogenic mechanisms of RA.^{2,3}

Objectives We have attempted to identify autoantigens that were recognised by newly identified autoantibodies discovered in RA patient sera. We hereby describe that human myc far upstream element binding protein (FUSEbp) is a new target of autoantibodies in RA.

Methods Molecular cloning was performed using a lambda gt11 cDNA library constructed from HeLa cell mRNA and RA patient sera that contain autoantibodies to unknown 75-kDa proteins as probes. cDNA inserts of isolated clones were sequenced, and detailed homologies of nucleotide and amino acid sequences were analysed through the BLAST server at NCBI DNA database. Immunoblot analysis was examined using bacterial lysates prepared from lysogenic E.coli Y1089 carrying the recombinant lambda phages. The partial cDNA (position 192–975 of FUSEbp

mRNA, termed FUSEbp-A) was amplified by PCR and ligated into an expression vector (pMAL-C2X). The expressed FUSEbp-A-maltose binding protein fusion protein was analysed by immunoblotting for patient sera.

Results The cDNA (termed RA9–2) expressing the fusion protein recognised by RA probe sera was isolated, and a 1.2 kb insert of RA9–2 was found to show a 99% homology at amino acid level with FUSEbp. The deduced molecular mass of FUSEbp was identical with the size of the 75-kDa protein in HeLa cell extracts. However, this expressed bacterial lysates containing the lambda phage were reactive with only one of 72 RA (1%) and one of 36 SLE (3%) patient sera. In contrast, the FUSEbp-A fusion protein was recognised by 7 of 72 RA sera (10%) as well as the probe serum in immunoblot analysis.

Conclusion Human myc FUSEbp, regulatory factor of c-myc expression,⁴ may be a novel autoantigen recognised by sera from patients with RA. In RA, synovial fibroblasts exhibit elevated gene expression of proto-oncogenes such as c-myc. Although it remains unclear how autoantibodies to FUSEbp can be associated with etiologic and pathogenic mechanisms in RA, increased expression and release of FUSEbp may induce autoantibodies to FUSEbp in RA-specific synovial proliferation and cartilage destruction.

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THU0064 CLINICAL SIGNIFICATION OF ANTI-KU ANTIBODY

¹F Limbach, ²J Goetz, ¹RM Javier, ¹JL Kuntz, ¹J Sibilia. ¹Rheumatology; ²Immunopathology, CHU Hautepierre, Strasbourg, France

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Background The Ku autoantigen is a DNA binding factor consisting of 70 and 80 kDa protein which form a heterodimer involved in repair of ds DNA breaks. Since the original description of anti-Ku antibody in patients with polymyositis-scleroderma overlap syndrome, this antibody has been showed to be associated with several autoimmune diseases.

Objectives To describe clinical features of 8 patients with anti-KU antibody.

Methods

Results

Conclusion

Abstract THU0064 Table 1

	Patient 1	2	3	4	5	6	7	8
Age (years)	28	40	44	30	66	41	67	74
ENA (ELISA)	Ku/Ro	Ku	Ku/RNP/JO1	Ku	Ku/proteinase3	Ku	Ku	Ku
Clinical features	Digital vasculitis	Sicca syndrome, polyarthritis	Myalgia, pulmonary fibrosis, sclerodactylia, raynaud's phenom	Raynaud's phenom	Extra-capillar glomerulonephritis, polyarthralgia, rectal carcinoma	Polyarthritis	Bronchopneumonia, Pulmonary embolism	Hand's oedema, prostatic cancer
Diagnosis	Lupus	Sjögren syndrome	Myositis-scleroderma	Raynaud	Wegener granulomatosis	Rheumatoid arthritis	Chronic obstructive lung disease	Prostatic cancer RS3PE ?