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

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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

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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 ?