Supplemental results

Additional patient characteristics

Patients with GCA had headache in 91% of the cases, scalp tenderness in 65%, jaw claudication in 66%, polymyalgia rheumatica according to Mayo Clinic Criteria (20, 25) in 53%, vision loss in 21% and a mean ESR of 68 mm/hour. Patients with APVA suffered from GPA in 7 of 10 cases and from microscopic polyangiitis (MPA) in 3 of 10 cases according to the definition of the Chapel Hill Consensus Conference (1). PR3-ANCA were found in 8 of 10 and myeloperoxidase ANCA in 3 of 10 patients. The median Birmingham activity vasculitis score was 8 (range 2–27). The median serum creatinine was 1.6 mg/dl. 45% of the patients with GPA, MPA or GCA were not treated by immunosuppressants at the time of sampling sera. Patients with rheumatoid arthritis (n=33) had a mean number of swollen joints of 3.3 (range 2–9 joints) and rheumatoid factor (>20 IU/ml) was present in 78% of patients. Extraarticular disease (three cases with major cutaneous vasculitis or neuropathy and one case each with pleuritis, pericarditis, keratoconjunctivitis sicca, Sjögren’s syndrome) was observed in 10/33 of patients (30%). For patients with Sjögren’s syndrome we found anti-nuclear antibodies (ANA) in 8/11 (73%), anti-Ro (anti-SSA) antibodies in 5/11 patients (45%) and anti-La (anti-SSB) antibodies in 4/11 (36%). 7/11 patients showed extraglandular disease becoming manifest in articular involvement (n=2), Raynaud’s phenomenon, autoimmune thyroiditis and renal involvement. Systemic vasculitis was present in 2/11 cases. In patients with SLE (n=23) ANA were found in 100% and anti-doublestranded DNA antibodies (anti-dsDNA) in 65% of the patients. The median SLEDAI was 9 (range 4–24). Systemic vasculitis was found in 3/23 patients becoming manifest in cerebral (n=2) or coronary vasculitis. In the control group, patients with cancer suffered from Hodgkin’s lymphoma, acute myeloid leukaemia, non-small cell lung cancer, pancreatic adenocarcinoma and renal cell carcinoma in 5 cases each.

References


Supplemental Fig. 1a.

Reactivity against lamin C detected by phage assay. Detection of serum reactivity against lamin C in a patient with giant cell arteritis (GCA) by phage assay. Phages from clones coding for lamin C were mixed with a phage clone (TTP2) of the same cDNA library as internal negative control at a ratio of 1: 10 and used to transfect bacteria. The 1: 100 diluted Escherichia coli-absorbed serum from a patient with GCA shows a positive reaction with the clone expressing lamin C in the phage assay while no reaction was observed with the clone which was used as control.

Supplemental Fig. 1b.

Evaluation of reactive clones by image analysis. Per nitrocellulose membrane 3 representative seroreactive phages and nearby non-reactive phages were selected. Staining intensity of the reactive plaque relative to the non-reactive plaque was calculated (relative density). For this purpose, a region of interest of the reactive plaque (grey box 1) or non-reactive plaque (black box 3) was chosen and region density was measured. This density was subtracted by the density of a region of background nearby the respective plaque (black box 2 for the reactive plaque and black box 4 for the non-reactive plaque). Relative density was calculated by the density of reactive plaques devided by the density of non-reactive plaques. A positive signal was defined as a signal with a relative density of at least 2.5 or more.