Objectives To evaluate safety, clinical improvement, and radiographic outcomes in patients receiving ENBREL or MTX for up to 2 years.

Methods Patients enrolled in this 2-year study continued to receive the therapy to which they had been randomised in an open-label fashion after the study was unblinded (at a mean of 17.3 months). The 2-year endpoints were ACR responses (using last on-drug observation carried forward analyses) and change in Sharp score. Readers were blinded to treatment and chronological order of the x-rays. All 632 patients were included in the analyses.

Results 154 of 207 patients (74%) who received ENBREL 25 mg completed 2 years of treatment, compared to 129 of 217 patients (59%) who received MTX. Twice as many patients (12%) discontinued MTX for adverse events as did those taking ENBREL. The ACR 20 response at 2 years was 72% in those randomised to ENBREL and 59% in those randomised to MTX ($P = 0.005$). Radiographic evaluations demonstrated that ENBREL 25 mg was superior to MTX in arresting radiographic progression over the 2-year period. Mean (median) changes in total Sharp score were 1.3 (0) and 3.2 (0.5) units in the ENBREL 25 mg and MTX groups, respectively ($P = 0.001$) and changes in erosion score were 0.7 (0) and 1.9 (0) units, respectively ($P = 0.001$). ENBREL 25 mg prevented radiographic progression in 63% of patients, compared to 51% of MTX patients who did not progress ($P = 0.017$). ENBREL 25 mg was also superior to the 10 mg dose in inhibiting radiographic progression. Significantly more ENBREL 25 mg patients than MTX patients had a clinically meaningful (at least 0.5 units) improvement from baseline in HAQ disability score. ENBREL continued to be well tolerated with over 2 years of exposure.

Conclusion This 2-year study demonstrated that ENBREL was superior to MTX in reducing clinical signs and symptoms of RA and inhibiting radiographic progression.

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SLE – Etiology and pathogenesis/Animal models

AB0069 ANTI BETA 2 GLYCOPROTEIN ANTIBODIES AND THROMBOSIS IN SLE PATIENTS

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Background

Objectives Beta 2 glycoprotein was identified as a cofactor for the binding of antiphospholipid antibodies. Anticardiolipin antibodies were found to correlate with thrombosis in SLE patients. We measured the subclasses of anti B2 GP1 in LSE patients with or without history of thrombosis.

Methods Fourty four SIE patients with mean ($\pm$ SD) age of 26.7 ± 9.9 years, nine of whom had a history of vascular thrombosis along with 30 healthy age and sex matched normal control subjects were studied.

Results There was a correlation between the presence of IgG anti B2 GP I and the incidence of thrombosis ($P < 0.05$). There was, also, a positive correlation between IgG anti B2 GPt and

IgG aCL antibodies and a negative correlation between IgG anti B2 GP1 and thrombocytopenia. However, serum levels of IgG and 1 gM anti B2GPI were increased in patients who had positive LAC activity. This thesis confirm the pathologic role of IgG anti B 2 GP1 (and not 1gM) in the incidence of thrombosis in SLE patients. Also, its relationship to the presence of anti ds DNA antibodies, anti CLA and lupus anticoagulant in SIE is discussed.

Conclusion

AB0070 THE EFFECT OF PROLACTIN ON DISEASE ACTIVITY IN SLE

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Background Prolactin, a hormone with immunomodulatory effects, possible plays a role in the disease activity of Systemic Lupus Erythematosus (SLE). Results of studies are contradictory regarding the correlation between hyperprolactinemia and disease activity.

Objectives To establish whether determination of prolactin is helpful in assessing disease activity in SLE by reviewing relevant literature.

Methods Studies were identified through Medline search. Also references from these articles were reviewed.

Results Ten articles that sought to establish a relationship between prolactin and SLE activity were identified.

Number of patients in the studies varied from 29 to 192, aged 16–73 years, and the percentage of elevated serum prolactin levels in SLE patients was between 2.2–40%. The SLEDAI was used as disease activity index in 7/10 studies. The presence of conditions known to be associated with hyperprolactinemia were either reported or excluded. Steroids treatment was accepted in all studies, DMARDs were used in 8/10 studies. In 5/10 studies bloodsamples were collected midmorning and in 4/10 patients had to fast. Single measurement of serum prolactin concentration and disease activity was done in 6 studies. The other 4 studies had 2–4 measurements per patient. Pearson's correlation coefficient was the used statistical analysis in 7/10 studies. A relation between prolactin and disease activity was reported in one study. The number of included patients in the studies was small and different statistical methods were used.

Conclusion The correlation between serum prolactin levels and disease activity is still uncertain. A cross-sectional as well as longitudinal study with using the correct statistics, power calculation and proper recording of secondary causes of elevated prolactin and medication need to be done.

FRI0083 ELEVATED IL-16 LEVEL IS A NON-GENETIC CHARACTERISTIC OF PATIENTS WITH SEVERE SYSTEMIC LUPUS ERYTHEMATOSUS

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Background IL-16, originilly named lymphocyte chemoattractant factor, is a cytokine which is mainly produced by CD8⁺ T cells. Several reports have described that increased levels of IL-

16 are in part responsible for T cell abnormalities in SLE patients.

Objectives To investigate whether increased levels of IL-16 is a characteristic underlying susceptibility to SLE or is a characteristic of the disease itself.

Methods Accumulated organ damage was measured with the SLICC/ACR Damage Index. Twenty-five severe (SLICC/ACR: 4.9 ± 2.5) and ten non-severe (SLICC/ACR: 1.0 ± 0.8) SLE patients were included in this study. Also 11 first degree relatives and 12 healthy volunteers were included in this study. Plasma IL-16 levels were measured by ELISA.

Results No significant difference in the IL-16 levels of the first degree relatives of patients with SLE (38.3 ± 11.1 pg/ml) were observed when compared to controls (31.2 ± 10.1 pg/ml). In order to analyse characteristics of the SLE in relation to concentration of IL-16, IL-16 was measured in severe SLE patients (71.3 ± 87.4 pg/ml; $P = 0.025$) compared to healthy controls. On the other hand, no significant differences were observed between the non-severe SLE patients (37.8 ± 26.1 pg/ml) and controls.

Conclusion No evidence for increased IL-16 levels in first degree relatives of the SLE patients was observed. IL-16 is enhanced in SLE patients with a severe disease, but not in patients with non-severe disease, thereby suggesting that IL-16 is associated with disease severity, and not with susceptibility for SLE.

FRI0084 AUTOPSY REPORTS OF THE CEREBRUM OF 24 PATIENTS WITH SLE IN THE PERIOD 1988–99

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Background Clinical manifestations of neurologic involvement in patients with SLE (Neuropsychiatric SLE or NPSLE) may be either due to secondary causes such as antiphospholipid-syndrome associated infarctions or bleedings or infections, or due to primary causes such as immune-mediated vasculitis or cerebritis. Depending on the probable cause, different therapeutic approaches are chosen.

Objectives Previous reports on histopathologic have suggested that, since it is so seldomly found, cerebral vasculitis cannot be an important cause of neurologic symptoms in SLE.

Methods

Results However, a search in the computer archives of PALGA, the foundation that collects all pathology reports from Dutch PA-laboratories, beginning in 1988 and covering all reports since 1990, revealed vasculitis lesions in no less than 5 of 24 cranial autopsies of patients with SLE. In an additional 4, lesions were described that were compatible with remnants of previous vasculitis.

Abstract FRI0084 Table 1 Conclusions of autopsy reports of 24 consecutive patients with SLE in the medical history and a cranial autopsy report

Primary NPSLE	7
Secondary NPSLE	9 (4 bleeding, 5 infection)
Other causes	6
No abnormalities/insufficient information	1
Insufficient information	1

Conclusion These findings suggest that cerebral vasculitis must be considered as a possible underlying cause of symptoms when it comes to choosing therapy for patients with NPSLE.

FRI0085 ANTI-BETA-2- GLYCOPROTEIN I ANTIBODIES IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background

Objectives

Methods Anticardiolipin antibodies (aCL) react with negatively charged phospholipids, which may often be complexed with a protein cofactor such as beta-2-glycoprotein I.

Results To obtain information about the prevalence and levels of IgG anti- β 2-GPI, IgG- and IgM-aCL in systemic lupus erythematosus (SLE) patients (pts) these antibodies were measured, by an enzyme linked immunosorbent assay, in sera from 147 pts with SLE (70 men and 77 women). The results were compared with 20 healthy subjects. Anti- β 2-GPI were found positive (more than 3s.d. above the mean in normal controls) in 15 females (19,4%), 14 males (20%) in pts with SLE compared to 1 person (5%) in normal controls. 48,3% of pts with anti- β 2-GPI had the history of thrombosis, compared with 11,02% of pts without anti- β 2-GPI ($p < 0,001$). 35,7% of females with anti- β 2-GPI had the history of fetal loss, in comparison with 6,56% of anti- β 2-GPI negative pts.

APS was diagnosed in 16 (69,56%) anti- β 2-GPI (+)-aCL(+) pts ($n = 23$) and in 15 (37,5%) anti- β 2-GPI (-)-aCL(+) pts ($n = 40$) ($p < 0,01$). Anti- β 2-GPI(+)-aCL(+) pts had a history of thrombosis more frequently ($n = 14$, 60,86%) than anti- β 2-GPI (+)-aCL(-) ($n = 6$) ($n = 0$, 0%, $p < 0,002$).

Our data show that the presence of anti- β 2-GPI simultaneously with aCL is strong associated with APS in SLE.

Conclusion

FRI0086 CHARACTERISATION OF RA33 (HNRNP-A2/B1)-AUTOREACTIVE T CELLS IN SLE-PATIENTS

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Background SLE is a systemic autoimmune disease with distinct immunological characteristics including defective T cell functions, especially concerning IL2 production and proliferation. Furthermore, B-cell hyperactivity is observed leading to the formation of several characteristic autoantibodies (ab), among them ab to the heterogenous nuclear ribonucleoprotein A2/B1 (hnRNP/RA33). These antibodies are known to occur in over 20% of SLE patients.

Objectives In order to elucidate the role of T cells and their influence in antibody production in SLE, we studied proliferation of PBMC to purified hnRNP-A2/B1 in SLE patients and healthy controls.

Methods Stimulation assays with PBMC of 34 SLE patients and 21 healthy controls were performed. We then proceeded to draw RA33-specific T cell clones (TCC) by cultivation and limiting-dilution cloning of T cell lines.