except for two died of miliary tuberculosis. A history of high dose steroid or cytotoxic drugs and any organ involvement were not related to the development of tuberculosis. SLE-associated tuberculosis was developed 48.13 $\pm$ 35.14 months after their initial symptom and 20.87 $\pm$ 23.53 months after clinical diagnosis.

Conclusion Taken together characteristics of tuberculosis in SLE patients are; 1) a higher incidence rate, 2) more frequent extrapulmonary involvements, 3) more extensive pulmonary involvements 4) a higher relapse rate (Odds ratio 4.14). Thus, the contributory role of M. tuberculosis infection in the morbidity and mortality of patients with SLE must be emphasised, especially in areas endemic for M. tuberculosis.

REFERENCES

- 1 Segal BH, Sneller MC. Infectious complications of immunosuppressive therapy in patients with rheumatic diseases. *Rheum Dis Clin North Am.* 1997;**23**:219–37
- 2 Petri M. Infection in systemic lupus erythematosus. *Rheum Dis Clin North Am.* 1998;**24**:423–56
- 3 Victorio-Navarra STG, Dy EER, Arroyo CG, Torralba TP. Tuberculosis among Filipino patients with systemic lupus erythematosus. *Semin Arthritis Rheum.* 1996;**26**:628–34
- 4 Kim HA, Yoo CD, Baek HJ, et al. Mycobacterium tuberculosis infection in a corticosteroid-treated rheumatic disease patient population. *Clin Exp Rheumatol.* 1998;**16**:9–13
- 5 Kim SJ, Hong YP, Lew WJ, Yang SC, Lee EG. Incidence of pulmonary tuberculosis in Korean civil servants. *Tuber Lung Dis.* 1995;**76**:534–9

FRI0099 CAUSES OF DEATH IN 56 PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Background

Objectives To describe the causes of death in a Chinese lupus cohort with SLE.

Methods We examined mortality in a cohort of 427 Chinese patients with SLE seen between 1985–1997. During a mean $\pm$ SD of 5.0 $\pm$ 3.8 years of follow-up, 56 patients died. The causes of death were determined.

Results The mean $\pm$ SD disease duration from diagnosis to last visit or death for the 427 patients was 6.7 $\pm$ 5.4 years, with a duration of follow-up of 5.0 $\pm$ 3.8 years. 53 females and 3 males died. The age at death was 35.3 $\pm$ 11.3 years. Among those who died, the duration of disease was 5.2 $\pm$ 4.8 years with a follow-up of 3.5 $\pm$ 3.3 years. In comparison, patients last seen alive had a longer disease duration of 6.9 $\pm$ 5.4 years and a longer follow-up of 5.3 $\pm$ 3.9 years. Infection and active SLE were the two leading causes of death, occurring in 17 patients (31%) in both instances. The clinical manifestations amongst the 17 patients with active SLE were as follows: seizure in 2, cerebritis in 1, cardiac tamponade in 2, pneumonitis in 2, pulmonary haemorrhage in 2, pulmonary hypertension in 2, nephritis in 2, vasculitis in 2, pancreatitis in 2. In 12 patients, SLE was a contributing factor of death. Thus, active SLE was present in over half of the cases (28 patients) at the time of deaths. The most common type of infection was pneumonia (41%) followed by bacteremias (35.3%). The most prevalent infections were bacterial infections (23.5%), followed by mixed infections (17.6%), fungal (17.6%) and mycobacterium infections (17.6%). Out of the 17 patients in whom the primary cause of death was due to infection, 9 had active SLE at the time of death.

Conclusion The majority of patients at the time of death had active SLE. Although the percentage of patients dying from infections and active SLE were the same, active SLE was present in half of those with infections suggesting that active disease remains the most important factor in mortality in SLE.

FRI0100 PREDNISONE (PRED), AZATHIOPRINE (AZA) AND CYCLOSPORIN A (CSA) COMBINATION IS EFFECTIVE FOR MEMBRANOUS LUPUS NEPHRITIS (MNL)

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Background

Objectives

Methods 10 consecutive female patients with mean age 35 $\pm$ 10 years and the mean disease duration prior to the onset of the index episode of nephritis was 86 $\pm$ 85 months with pure MLN were treated with prednisolone, AZA and CSA for at least 12 months. CSA was tapered in stable patients by 18–24 months. All patients were followed for 3 years.

Results At 1 year, 4 patients achieved complete response as defined as serum albumin over 35 g/L, urinary protein less than 0.3 g/day and serum creatinine less than 30% above the baseline value and 3 achieved partial response. One patient developed agranulocytosis and alopecia totalis, another patient withdrawn at 6 months because of inefficacy, and one patient had no response at 1 year. For the responders, serum albumin increased from 22.8 $\pm$ 5.1 to 34.4 $\pm$ 2.7 mg/l, accompanied by reduction in urine protein from 5.6 $\pm$ 3.2 to 0.8 $\pm$ 0.8 g/day, while serum creatinine remained unchanged. Significant increase in serum albumin and sustained reduction in urine protein were noted from 3 months onward. Creatinine clearance showed significant improvement by the end of 1 year. Serum C3 normalised in 3 out of 6 patients, whereas anti – ds DNA antibodies became negative in 5 out of 6 patients. The mean prednisone dose reduced from 26.8 $\pm$ 5.5 to 5.7 $\pm$ 1.9 mg/day, the mean dose of CSA also reduced from 235.7 $\pm$ 24.4 to 132.1 $\pm$ 49.4 mg/day at the end of 1 year.

Side effects included hypertension requiring additional anti-hypertensives in 4 patients, minor infections in 2 patients and leucopenia in 1 patient. CSA was tapered by the end of second year in 4 patients. At 3 years, 6 patients had sustained response, 2 patients developed renal relapse at 18 and 20 months. One patient reached end stage renal failure at 27 months.

Conclusion Combination therapy using prednisolone, AZA and CSA is well tolerated and is efficacious in the management of MLN, and may prevent relapse in patients after stopping CSA.

FRI0101 ANTIRIBOSOMAL-P PROTEIN ANTIBODIES IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background Autoantibodies to ribosomal-P protein (anti-P antibodies) are predominantly found in sera of patients with systemic lupus erythematosus (SLE) and have been correlated with neuropsychiatric lupus in some but not all studies.¹ The