appropriate evaluation of synovial fluid be carried out in all such patients.

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References

- 1 Rothschild B M, Sienknecht C W, Kaplan S B, Spindler J S. Sickle cell disease associated with uric acid deposition disease. *Ann Rheum Dis* 1980; **39**: 392-5.
- 2 Ballou S P, Khan M A, Kushner I, Harris J W. Secondary gout in hemoglobinopathies: report of two cases and review of the literature. *Am J Hematol* 1977; **2**: 397-402.
- 3 Crosby W H. The metabolism of hemoglobin and bile pigment in hemolytic disease. *Am J Med* 1955; **18**: 112-22.
- 4 Gold M S, Williams J C, Spivack M, Graun J. Sickle cell anemia and hyperuricemia. *JAMA* 1968; **206**: 1572-3.

Acquired antithrombin III deficiency and systemic lupus erythematosus

Sir,

We read with interest the report by Gladman and Urowitz on thromboembolic disease and systemic lupus erythematosus (SLE).¹ The thrombotic diathesis in this group of patients was, however, not explained. It has been well established that familial antithrombin III deficiency is associated with a thrombotic diathesis.² Kauffman and coworkers have linked an acquired antithrombin III deficiency in patients with the nephrotic syndrome to their development of renal vein thrombosis and extrarenal sites of thrombosis.³ They demonstrated a highly significant negative correlation between serum antithrombin III concentration and urinary protein excretion ($r = -0.58$, p 2-sided <0.001). Their findings suggested that the low antithrombin III levels were due to loss of the molecule in the urine. Because renal disease with proteinuria is a frequent complication of SLE, we decided to determine plasma antithrombin III levels in a group of SLE patients.

Plasma antithrombin III concentration was assayed by radial immunodiffusion (Behring Diagnostic Corp., La Jolla, CA) on 33 plasma samples from 27 patients with documented SLE, i.e., meeting at least 4 ARA criteria for SLE.⁴ Twenty-four hour urine collections for creatinine and protein excretion were obtained on all patients within 1 week of the plasma sample. Creatinine clearances in all patients studied were greater than 30 ml/minute (mean = 68 ml/minute). Protein excretion ranged from 0 to 24.13 g per 24 hours (mean = 3.15 g per 24 hours).

There was a significant inverse correlation of antithrombin III level and urinary protein excretion ($r = -0.413$, p 2-sided <0.05). Antithrombin III levels were below normal in 5 patients, on at least 1 determination. Four of these patients had a rise in antithrombin III level to normal on serial determinations. In 2 of these patients there was a simultaneous dramatic decrease in urinary protein excretion of at least 6 g of protein per 24

hours. In the other 2 patients urinary protein excretion did not change during the period that their antithrombin III levels normalised. Both of these patients had clinical and serological evidence of active SLE at the time their antithrombin levels were low. Follow-up samples during disease remission showed normal antithrombin III levels. During the study period thromboembolic disease was not documented in any of the patients.

In conclusion, we have demonstrated that patients with SLE can have an acquired antithrombin III deficiency. There is a significant inverse correlation of antithrombin III level and urinary protein excretion. However, the normalisation of the antithrombin III levels in 2 patients occurred when their SLE became inactive, suggesting that low antithrombin III levels in SLE patients may be related to factors other than urinary protein excretion. Only a long-term prospective study of SLE patients with serial antithrombin III assays will determine if there is a relationship between and acquired antithrombin III deficiency and thromboembolic disease in SLE.

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References

- 1 Gladman D D, Urowitz M B. Venous syndromes and pulmonary embolism in systemic lupus erythematosus. *Ann Rheum Dis* 1980; **39**: 340-3.
- 2 Marciniak K E, Farley C H, DeSimone P A. Familial thrombosis due to antithrombin III deficiency. *Blood* 1974; **43**: 219-30.
- 3 Kauffman R H, Velkamp J J, Van Tilburg N H, Van Es L A. Acquired antithrombin III deficiency and thrombosis in the nephrotic syndrome. *Am J Med* 1978; **65**: 607-13.
- 4 Cohen A S, Reynolds W E, Franklin E C, et al. Preliminary criteria for the classification of systemic lupus erythematosus. *Bull Rheum Dis* 1971; **21**: 643-6.

Beta-2-microglobulin in RA

Sir, In 2 recent studies on serum beta-2-microglobulin ($\beta_2\mu$) in rheumatoid arthritis (RA)^{1,2} the results are somewhat divergent with regard to the correlation between $\beta_2\mu$ and the disease activity. We have studied the same subject and would like to report our findings.

Our material consisted of 51 consecutive inpatients with classical RA (ARA criteria 1958). One patient was excluded because of paraproteinaemia.

The clinical examination showed that the 50 patients could be separated into 2 distinct groups with regard to the disease activity. In 39 patients (mean age 53.8 ± 12.0 years, range 25-72 years) no more than a few joints were actually inflamed. Their disease was considered