Dear Sir,

CRP is one of the most sensitive acute-phase reactants [4]. This protein seems to play a role in the regulation of platelet function, by acting through the inhibition of platelet aggregation. Platelet factor 3 activation by heat-aggregated $\gamma$-globulins and platelet aggregation by thrombin, ADP, collagen have been reported to be blocked by CRP in vitro [2].

In nephrotic syndrome, hemostatic abnormalities have been described, with a tendency towards hyper-coagulability [3]. These data led us to investigate CRP values and some hemostatic parameters [6] in 6 rheumatoid patients who suffered from nephrotic syndrome while on Tiopronin treatment (a sulphydryl compound similar to D-penicillamine) [1]. The results of laboratory investigation and thromboelastography data [6] are shown in table I.

Two main findings did arise from this survey:

1. CRP values were within normal range in 5 of 6 patients and a complete dissociation between CRP and ESR was observed. Therefore the nephrotic syndrome is one of the few clinical conditions exhibiting such a discrepancy [4]. The low values of CRP very likely depend on the renal loss of the low molecular weight protein, as described for other molecules [5].

2. A tendency towards hypercoagulability was observed in 5 of 6 patients. More specifically, very high levels of fibrinogen (over 700 mg/dl) and an increased maximum amplitude were found by thromboelastography in 5 of 6 patients. Platelet count and prothrombin activity were normal. Hematocrit was low in a single patient.

Owing to the claimed anticoagulant activity, the low levels of this protein could represent a contributing factor to the development of a hypercoagulability state, as observed in our nephrotic patients.

References

Table I. ESR, CRP and hemostatic parameters recorded in 6 rheumatoid patients with nephrotic syndrome due to Tiopronin

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