CRP in Nephrotic Syndrome: Relationship with Hemostatic Abnormalities?

G.F.		Ferraccioli
F.		Cavalieri
M.		Mercadanti

Rheumatic Disease Unit, University of Parma, Parma, Italy

G.F. Ferraccioli, MD, Rheumatic Disease Unit, University of Parma, Via Gramsci 14, I-43100 Parma (Italy)

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 γ -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 sulphhydryl 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

1 Ferraccioli, G.F.; Cavalieri, F.; Mercadanti, M.; et al.: Clinical features, scintiscan characteristics and X-ray progression of late onset rheumatoid arthritis. Clin. exp. Rheumatol. 2: 157-161 (1984).

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

Fiedel, B.A.; Gewurz, H.: Effect of C-reactive protein on platelet function. I. Inhibition of platelet aggregation and release reactions. *J. Immun.* 116:1289–1295 (1976).

Panicucci, F.; Sagripanti, A.; Vispi M.; et al.: Comprehensive study of hemostasis in nephrotic syndrome. *Nephron* 3: 9–13 (1983).

Pepys, M.B.: C-reactive protein: the role of an ancient protein in modern rheumatology. *Clin. exp. Rheumatol.* 1:3–8 (1983).

Yatzidis
H.; Richet
G.: Activité de certaines protéines de la coagulation dans le plasma et les urines au cours du syndrome néphrotique. *Rev. fr. Etud. clin. bio.* 2:717–727 (1957).

6 Zuckerman
L.; Cohen
E.; Vagher
J.F.; et al.: Comparison of thromboelastography with common coagulation tests. *Thromb. Hemostasis* 46:752–756 (1981).