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LETTER TO THE EDITOR

İ. C. Haznedaroğlu · Y. Büyükaşık · Y. Erdem

Increased thrombin and plasmin generation in chronic renal failure

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Renal disease can result in significant disorders of hemostasis. Patients with end-stage renal disease have increased morbidity and mortality from thrombosis, and yet paradoxically may have a hemorrhagic diathesis. Both types of coagulopathies may occur in the same patient. The bleeding tendency generally results in mucosal bleeding and increased blood loss with surgical procedures. The hypercoagulability state leads to thrombotic events, such as myocardial infarction, pulmonary embolism, renal vein thrombosis, and thrombosis of arteriovenous fistula (AVF). We have read with great interest the article by Sagripanti et al. [1], demonstrating an increased concentration of prothrombin fragment 1+2, the peptide fragment generated when prothrombin is activated to thrombin, in uremia. These findings confirmed our previous investigations which indicated that hemostatic markers of procoagulant imbalance are increased in hemodialysis patients [2–6]. We also demonstrated increased fibrinolysis in plasma and AVF of hemodialyis patients, supporting the idea that both coagulation and fibrinolysis are simultaneously activated in chronic renal failure as "*two cats in a bag*" [3–7]. Since normal hemostasis depends on a balance between the procoagulant system with associated inhibitors and the fibrinolytic system and its inhibitors, this may explain the thrombotic tendency of bleeding-prone uremic patients.

Systemic amyloidosis is also known to be frequently associated with pathological hemostatic plug formation and might be an additional factor causing bleeding tendency in patients on hemodialysis programs. Accelerated fibrinolysis was implicated in this pathological condition and greatly increased plasmin-α_2 antiplasmin complex, which is a molecular marker of the fibrinolytic system, was found in plasma and AVF of amyloid patients in our recent studies [5, 6]. Finally, we demonstrated the role of AVF in hemostatic defects observed in patients with end-stage renal disease on maintenance hemodialysis [4, 5, 7]. Changes in vessel wall and/or blood flow in native AVF might increase hemostatic disarrangements, as vascular endothelium is predominantly involved in the regulation of hemostatic pathways. Much additional work is still necessary to clarify the activation inhibitory mechanisms of coagulation and fibrinolysis in uremic patients. It is hoped that future studies will shed further light on these issues.

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İ. C. Haznedaroğlu (✉)
36. Sok. Sun apt. No. 3/2, TR-06490 Bahçelievler, Ankara, Turkey

I. C. Haznedaroğlu · Y. Büyükaşik · Y. Erdem
Departments of Nephrology and Hematology,
Hacettepe University School of Medicine, Ankara, Turkey