Dear Sir,

In a recent article by Nomura et al. [1] a reduction of platelet glycoprotein GPIb expression in the uremic patient was confirmed, just as reported by Sloand et al. [2]. We agree with these data, that clarify the important role of a defect in primary hemostasis in the pathogenesis of uremic bleeding. We investigated the abnormalities of platelet surface glycoproteins GPIb, the receptor for von Willebrand factor, and GPIIb/IIIa, the receptor for fibrinogen, since the early stages of renal failure and we correlated them with the degree of uremia and plasma creatinine. We studied four groups of patients: group A (n = 18 healthy controls) creatinine = 0.8 ± 0.3 mg%; group B (n = 10) creatinine = 1.8 ± 0.5 mg%; group C (n = 8) creatinine = 5.4 ± 2.1 mg%; group D (n = 10) creatinine = 9.6 ± 3. In all the patients we investigated platelet glycoproteins GPIb and GPIIb/IIIa with monoclonal antibodies CD42b and CD41 (Biorad, Italy) and flow cytometric analysis (Bryte cytometer; Biorad). Mean values of GPIb glycoproteins (mean flow ± SD): group A = 43.09 ± 11.19; group B = 37.84 ± 5.57; group C = 34.82 ± 9.54 (p < 0.05); group D = 33.02 ± 7.6 (p < 0.05). Mean values of GPIIb/IIIa: group A = 312.40 ± 108.3; group B = 410.63 ± 34.65 (p < 0.05); group C = 434.89 ± 50.86 (p < 0.025); group D = 415.31 ± 70.5 (p < 0.05). Our data suggest: (1) that platelet surface glycoprotein abnormalities are present since the early stages of renal failure and are well correlated with the degree of uremia; (2) that also glycoprotein GPIIb/IIIa is altered in uremia (these further data are different from the data reported by other authors), and (3) that the defect is not corrected by dialytic procedure. Finally, we want to stress in uremic patients the main importance of platelet glycoprotein abnormalities that also have an important role in the abnormal procoagulant response to treatment with recombinant human erythropoietin, as just reported [3].

References