Treatment of Gross Hematuria in Autosomal Dominant Polycystic Kidney Disease with Aprotinin and Desmopressin Acetate

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Sir,

Gross hematuria as a result of cyst bleeding is a common complication in autosomal dominant polycystic kidney disease (ADPKD) [1]. Local activation of fibrinolysis by urokinase may exaggerate bleeding, therefore cessation of bleeding is often prolonged or yet impossible.

In the following, we report on a 30-year-old male with ADPKD. A life-threatening gross hematuria developed after strenuous physical exercise. The patient was admitted to our ward with gross hematuria and right-sided loin pain. Upon sonography, 2 renal cysts (4.5- and 3-cm diameter) could be identified and were suspected to be the source of renal bleeding. There was no evidence of obstruction along the urinary tract. Laboratory investigations revealed no abnormalities in renal function (s creatinine 1.4 mg/dl, urea 24 mg/dl) and hemostasis (plasmatic coagulation: prothrombin time, aPTT, thrombin time, F VIII). Platelet function (bleeding time, platelet aggregation tests: ADP, collagen, ris-tocetin, arachidonic acid) was also within the normal range.

Severe anemia had developed (hemoglobin decreased from 14 to 9 g/dl), the calculated daily blood loss (urocrit) was 375 ml. Blood transfusion of 2 U every other day (total: 20 during the clinical course) were required to stabilize hemoglobin levels at 8 g/dl. An infusion with aprotinin (Trasylol Bayer, FRG) and desmopressin acetate (DDAVP) 0.4 µg/kg was initiated every other day. Hematuria decreased during infusion of DDAVP, but increased immediately after the end of the infusion.

Aprotinin was added in increasing doses, starting with a daily dosage of 100,000 kallikrein inhibitor units (KIU) after allergic reactions to the drug were excluded. Bleeding ceased after a 3-day course with high doses of aprotinin (1.2 mil KIU/day), without any renal obstructive or thrombotic complications.

The present case shows the effectiveness of aprotinin in the management of severe renal bleeding and the superiority over DDAVP.

The successful administration of aprotinin in bleeding complications after cardiopulmonary bypass [2] or in vascular surgery [3] has been reported previously.

A treatment of renal bleeding with antifibrinolytic agents is also documented by the use of epsilon aminocaproic acid (EACA) [4]. In view of the high urinary concentration of EACA, the danger of obstructive clot formation in the urine is increased [5]. In contrast, aprotinin is only excreted in minor amounts in urine and to our knowledge, there is no single case of urogenital clot formation reported. In our opinion, the administration of aprotinin in renal bleeding is appropriate and can prevent invasive measures.

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