Acute Renal Failure Secondary to Antifibrinolytic Therapy

Letter to the Editor

Nephron 1995;69:478-479

M. Fernández Lucas, MD, Department of Nephrology, Hospital Ramón y Cajal, Crta. de Colmenar, Km 9.100, E-28034 Madrid (Spain)

Department of Nephrology and Hematology, Hospital Ramón y Cajal, Madrid, Spain

Dear Sir,

Antifibrinolytic agents have been claimed to be clinically efficient in patients with a generalized bleeding disorder such as hemophilia or chronic severe thrombocytopenia [1]. However, this therapy is not without risks, and intrarenal obstruction has been described as a complication of antifibrinolytic drugs in hemophiliacs [2, 3]. We report a patient with a diagnosis of myelodysplastic syndrome (refractory anemia with excess of blasts) and severe thrombocytopenia, who was treated with prophylactic doses of tranexamic acid and developed acute renal failure. To our knowledge, this is the first case described in the literature.

Myelodysplastic syndrome was diagnosed in a 66-year-old man in December 1992. He was treated with cycles of low-dose cytarabine and granulocyte colony-stimulating factor between February and May 1993, and low doses of tranexamic acid when platelet count was below 50,000/mm3. The patient was admitted on May 30 due to edema, oliguria and bilateral lumbar pain. Before admission, he had been treated with 1 g/6 h of tranexamic acid for 15 days. Laboratory investigations showed: serum creatinine 7mg/dl (619 mol/l), BUN 69 mg/dl (24.6 mol/l), creatinine clearance 5 ml/min/1.73 m2, hemoglobin 8 g/dl, hematocrit 26%, white cells 8,100/mm3 (77% neutrophils, 13% lymphocytes, 9% monocytes), platelets 30,000/mm3. Prothrombin activity and partial thromboplastin time were normal. Urine output was 300 ml/24 h with macroscopic hematuria and clots. The following data were normal or negative: immunoglobulins (IgG, IgA, IgM), complement (C3, C4, CH50), rheumatoid factor, antinuclear antibodies, cryoglobulins, c-ANCA, p-ANCA and urine and blood cultures. Renal ultrasonography showed moderate bilateral hydronephrosis with slightly enlarged kidneys. Radioisotope renal scanning showed a normal bilateral renogram. A retrograde ureterography showed filling defects in the pelvis and in the calyceal system of the left kidney. The right kidney showed a calyceal diverticulum.

© 1995 S. Karger AG, Basel
0028-2766/95/0694-0478
$8.00/0

Fig. 1. Retrograde pyelography showing filling defects in the pelvis and in the calyceal system of the left kidney. The right kidney shows a calyceal diverticulum.

Fig. 2. Retrograde pyelography showing filling defects in the pelvis of the right kidney.
teropyelogram showed bilateral filling defects within the pelvis and the calyceal system (fig. 1, 2). Multiple clots and fibrin deposition were found in the bladder and in the ureteric orifices at cystoscopy. The patient was in anuria for 15 days and needed hemodialysis. When diuresis was recovered, the urine showed small brown clots. He was discharged 1 month later in good general condition, but with an impaired renal function (creatinine clearance 33 ml/min/1.73 m2).

The antifibrinolytic effect of tranexamic acid is related to reversible complex formation with a modified plasminogen [1]. This complex blocks the interaction between plasminogen and fibrin which results in delayed fibrinolysis [1]. This agent has been used as a prophylactic drug in patients with high risk of bleeding.

The retrograde pyelography in our patient suggested that intrarenal obstruction was due to blood clots in the renal pelvis, although Stark et al. [2] described intrarenal obstruction by intratubular fibrin deposition in hemophiliacs treated with antifibrinolytic drugs. As spontaneous lysis of the intrarenal clots occurs within 2-3 weeks, no specific measures are required. However, acute renal failure is a potential severe complication of antifibrinolytic therapy and this should be remembered when these agents are indicated.

References
Marder VJ, Butler FO, Barlow GH: Antifibrinolytic therapy; in Colman RH (ed): Hæmoto-