Tranexamic-acid-induced acute renal cortical necrosis in a patient with haemophilia A

Sir,

Recently, Koo *et al*. [1] reported about a patient with acute renal cortical necrosis caused by antifibrinolytic drug (tranexamic acid) in this journal. We observed a very similar case, where a patient with haemophilia A was treated with an antifibrinolytic drug (tranexamic acid) and developed acute renal cortical necrosis.

Case. A 21-year-old man was admitted to our department because of oligoanuria. Since childhood the patient had suffered from intra-articular bleeding after minor trauma and epistaxis which led to the diagnosis of haemophilia A at the age of 5 years. He was well until 3 days prior admission, when he had a new epistaxis attack. Before the current admission to our department the patient was first admitted to another hospital, where tranexamic acid, 3 g/day for 4 days, was administered. However, 3 days later the patients suddenly developed oligoanuria and azotaemia and was therefore transferred to our department. On examination, the patient's epistaxis was under control. The urinary analysis was normal (no proteinuria and no haematuria), haemoglobin 13.6 g/dl, BUN 54 mg/dl, and serum creatinine 3.8 mg/dl. ANA, anti dsDNA, and ANCA were all negative or normal. Except for PTT, the coagulation profile was normal. Renal ultrasonography and echocardiography showed normal findings.

On the sixth day of hospitalization, the patient's serum creatinine increased to 8.1 mg/dl. He was treated by haemodialysis for 3 weeks. During follow-up, urine output decreased below 20 cc/day and did not increase again. Renal angiography was performed and diminished cortical contrast enhancement was observed, compatible with the diagnosis of renal cortical necrosis.

Comment. The incidence of renal cortical necrosis is highest in obstetrical patients, especially in late pregnancy. It is also seen as part of the haemolytic–uraemic syndrome, significant haemorrhage, toxaemia, disseminated intravascular coagulation, severe hypotension, some drugs, and following ethylene glycol or alcohol poisoning [2]. Our patient was young and none of the above-mentioned reasons was present. Urinary analysis showed no haematuria or proteinuria. There was no history of hypotensive episodes and none of the clinical and laboratory findings of microangiopathic haemolytic anaemia was present.

Renal impairment associated with acute renal cortical necrosis caused by tranexamic acid is rare. Only three cases have previously been recorded in the English literature. In these cases it was reported that intraglomerular capillary and arteriolar fibrin thrombosis with infarcts developed together with the use of tranexamic acid [1,3,4]. Two of these three cases had risk factors for ischaemic renal damage and acute renal cortical necrosis such as hypotension and advanced age [3,4]. In the third patient there were no known risk factors for acute renal cortical necrosis [1]. In our patient the bilateral acute renal cortical necrosis was also seen in the absence of classical risk factors for ischaemic acute renal failure.

The definitive diagnosis usually requires renal biopsy. Renal angiography can also be used since it can reveal characteristic findings [5]. We made the diagnosis of bilateral acute renal cortical necrosis with renal angiography. Due to development of acute renal cortical necrosis in association with the use of tranexamic acid, we recommend that this drug be prescribed only on the basis of appropriate indications. When used, all its potential complications should be considered.

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