Renal Infarction in a Patient without Underlying Diseases

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Dear Sir,

Renal infarction usually occurs in patients with severe atherosclerosis and heart diseases, but it rarely occurs in a patient without such underlying diseases [1]. We report on a 46-year-old woman who developed left renal infarction, having no underlying diseases.

On November 4, 1992, the patient felt fever, consulted a nearby doctor, and received 25-50 mg/day of diclofenac, a non-steroidal anti-inflammatory drug (NSAID). At this time, the serum creatinine level was 0.7 mg/dl. About 1 week later, she was admitted to a nearby hospital for the development of generalized edema and weight gain of 4 kg within 1 week. A tentative diagnosis of acute glomerulonephritis was made. Diclofenac was discontinued. The blood pressure was 170/100 mm Hg. Protein and occult blood in the urine were detected, but proteinuria was undetectable 3 days later. Hypertension and edema were initially treated by diuretics (furosemide 40 mg/day and spironolactone 50 mg/day) and thereafter in combination with an angiotensin-con-verting enzyme inhibitor (captopril 37.5 mg/ day). On November 20 and 21, she received 25 mg of diclofenac for the treatment of headache. On November 22, she complained of nausea and left-flank pain; laboratory investigation revealed hematuria, proteinuria, and an increase of the serum creatinine level to 1.8 mg/dl.

On November 25, 1992, she was transferred to our hospital for further evaluation of the renal function. On the day of admission physical examination revealed fever of 37.8°C and left lower abdominal tenderness. No pitting edema was observed on her extremities. The blood pressure was 132/ 80 mm Hg. Urinalysis disclosed 4+ hematuria, 3+ proteinuria, and numerous red blood cells. The hemoglobin level was 10.7 g/dl, the white blood cell count 18,800/ μ l, the platelet count $23.6 \times 10^4/\mu$ l, and the C-reactive protein level was 16.2 mg/dl. A coagulation test was normal. The blood urea nitrogen level was 13.6 mg/dl, serum creatinine 0.80 mg/ dl, aspartate aminotransferase 40 IU/l, ala-nine aminotransferase 48, and the lactate dehydrogenase level was 1,917 IU/l. The serum albumin concentration was normal. Repeated urinalysis performed 2 days later showed no proteinuria, but 10-15 red blood cells/high-power field. Negative serological tests for antinuclear antibody, rheumatoid factor, anti-DNA antibody, and antistreptolysin O titer were obtained. The serum complement levels were normal.

A computerized tomography scan of the abdomen performed using contrast medium on December 7 revealed wedge-shaped low-density lesions in the left kidney, suggestive of segmental infarction. No anticoagulation therapy was instituted because the renal function, on

the basis of the serum creatinine levels, was normal. Left renal angiography performed on December 22, 1992, did not reveal definite arterial thromboembolism, but showed segmental hypoperfusion due to narrowed arterial branches, resulting in defects present in the nephrogram with focal cortical atrophy at the relevant sites. It also demonstrated retrograde filling of the gonad-

Fig. 1. Venous phase of the renal angiography. The defects with focal atrophy are shown, and the gonadal vein is opacified, suggestive of nutcracker phenomenon.

al vein, suggestive of nutcracker phenomenon (fig. 1).

Thromboembolism to the renal arteries occurs most often in patients with underlying diseases that have the potential for mural thrombus formation [1]. Our patient developed unilateral renal infarction despite the absence of underlying diseases. The lack of underlying diseases led to the suggestion of an unusual mechanism involved in our patient. Our patient presented with edema, hypertension, and impaired renal function with transient proteinuria at a nearby hospital. These symptoms appeared to be related

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to the diverse effects of NSAIDs such as diclofenac because there were no findings suggestive of acute glomerulonephritis or collagen diseases. Acute renal failure with flank pain and/or patchy renal vasoconstriction associated with NSAIDs have been reported [2]. We also reported acute renal failure due to patchy vasoconstriction associated with rhabdomyolysis [3], hypotension [4], and analgesics used in a cirrhotic patient [5]. Diclofenac in this patient appears to have induced acute renal failure which resulted in an increase in plasma volume, leading to generalized edema and high blood pressure, probably associated with the development of patchy renal vasoconstriction. It is suggested by the narrowing of the renal arterial branches observed by renal angiography without abrupt cutoff signs. Renal perfusion in the areas affected by vasoconstriction may have been maintained by increased plasma volume and high blood pressure, but eventually reduced by the treatment with diuretics and captopril. In this situation additionally given diclofenac and nutcracker phenomenon involved in our patient may have worsened renal blood flow and venous return in the left kidney, leading to the ischemia and subsequent necrosis in the most severely damaged regions.

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