Wedge-Shaped Low-Density Lesion on Sonography of the Kidney in Advanced Cirrhotic Patient

T.	Takanobu	Sakemi
M.	Masako	Kikuchi
S.	Sumiko	Takahashi

Departments of Internal Medicine and Radiology, Saga Medical School, Saga, Japan

Takanobu Sakemi, MD, Department of Internal Medicine, Saga Medical School, Nabeshima, Saga 849 (Japan)

Dear Sir,

Progressive oliguric renal failure commonly complicates the course of patients with advanced cirrhosis. Cirrhotic patients have been reported to show a characteristic renal circulatory disturbance, such as renal vasoc-onstriction leading to severely reduced renal blood flow and reduced glomerular filtration rate. We report on a cirrhotic patient who developed acute renal failure (ARF), associated with the occurrence of lower back pain and wedge-shaped low-density area on sonography of the kidney.

A 57-year-old woman was admitted to another hospital because of further examination of ascites on June 19, 1989. She was diagnosed as having advanced liver cirrhosis with ascites and esophageal varices. On July 12, she developed acute cholecystitis, demonstrating a fever of 40 °C and a jaundice and received antipyretics for 3 days and antibiotics for 5 days. Two days later, she noticed lower back pain and macrohematuria, followed by oliguria and rapid increase in serum creatinine and thereby underwent hemodialysis (HD).

She was transferred to our hospital because of further examination of ARF on July 25 (the 12th clinical day). Physical examination on admission revealed distended abdomen with tenderness and fluctuation. Blood pressure was 112/52 mm Hg. There was a dull pain over the bilateral costovertebral angles. There was no pitting edema on her extremities. Urine volume was < 100 ml per day. Protein and occult blood in urine were positively detected; the sediment contained numerous red blood cells and many hyaline and granular casts. Urine sodium was 74 mEq/l and potassium 30.5 mEq/l. Fractional excretion of urinary sodium (FENa) was 3.9%. The hemoglobin level was 7.4 g/dl; white blood cell count 1,700/μl and platelet count 45,000/μl. Blood urea nitrogens was 44.9 mg/dl and serum creatinine 8.1 mg/dl. Total bilirubin was 1.3 mg/dl, aspartate aminotransferase 24 U/l, alanine aminotransferase 8 U/l. Total protein was 6.2 g/dl, albumin 3.3 g/dl, IgG 2,468 mg/dl, IgA 138 mg/dl and IgM 282 mg/dl. HD was performed 9 times during the following 3 weeks and discontinued thereafter because of the increase in urine output and fall in serum creatinine. The first renal sonography performed on the second hospital day revealed enlarged kidneys with the wedge-shaped low-density area in the left

Fig. 1. The first renal sonography: wedge-shaped low-density lesion is seen in the middle of the left kidney.

one, but on the second renal sonography which was performed 14 days later, when urine output gradually increased up to 600 ml/day under receiving HD treatment, this wedge-shaped lesion disappeared.

118

Sakemi/Kikuchi/Takahashi

Renal function rapidly deteriorated, following cholecystitis-associated fever and jaundice and treatment with antipyretics and antibiotics, to the level of requiring hemodialysis, with accompanying symptoms of macroscopic hematuria, oliguria and lower back pain. Acute renal failure in this patient is suggested to be due to acute tubular necrosis (ATN) on the basis of the findings of high urine sodium concentration, high FENa, urine sediment and clinical course. Disappearance of wedge-shaped lesion disclosed on the second sonography excluded the possibility of renal infarct.

The occurrence of ATN in patients with cirrhosis may relate to the hypotension, infection, jaundice and multiple metabolic disorders that complicate the course of the patient. Cirrhotic patients have been reported to tend to develop renal vasoconstriction [1]. Vasodilator prostaglandins induce a pivotal protective compensatory vaso-dilation in setting of renal hypoperfusion, when activation of the renin-angiotensin cascade alone would threaten the kidney with further vasodilation, such as advanced cirrhosis [2]. In this situation, inhibitors of prostaglandin synthesis produce acute deterioration of renal blood flow and glomerular filtration rate. It is conceivable that infection and associated jaundice predisposed our patient to ATN and in turn prostaglandin inhibitor broke the increase in angiotensin-dependent prostaglandin synthesis, leading to renal vasoconstriction and resultant overt ATN.

Wedge-shaped lesion on computed tomography which was regarded as indicative of patchy vasoconstriction, has been reported in the patients with acute renal failure due to nonsteroidal antipyretic drug [3], rhabdomyolysis [4] or hypotension [5]. We regarded wedge-shaped lesion of renal sonography in our case as the same kind of nature in origin as those of previous reports and considered its participation in occurring of ATN. Our case suggests that reduction of renal blood flow and glomerular filtration rate followed by use of nonsteroidal antipyretic drugs may be associated with patchy renal vasoconstriction.

References

Epstein M: Liver disease; in Massry SG, Glasscock RJ (eds): Textbook of Nephrology. Baltimore, Williams & Wilkins, 1989, vol 2, pp 966–977.

Levenson DJ, Simmons CE Jr, Brenner BM: Arachidonic acid metabolism, prostaglandins and the kidney. *Am J Med* 1982; 72:354–374.

Ishikawa I, Saito Y, Shinoda A, et al: Evidence for patchy renal vasoconstriction in man: Observation by CT scan. *Nephron* 1981;27:31–34.

Ozaki I, Sakemi T, Sanai T, et al: Patchy renal vasoconstriction in rhabdomyolysis-related acute renal failure. *Nephron* 1988; 48:136–137.

Sakemi T

Kudo S

Nagano Y

et al: Persistent wedge-shaped low-density lesions on computed tomography of the kidneys without infarction. *Nephron* 1989;51:112–114.