Persistent Wedge-Shaped Low-Density Lesions on Renal Sonography

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Dear Sir,

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 [1], rhabdomyolysis [2] or hypotension [3]. Wedge-shaped lesion on renal sonography has rarely been reported [4]. We report on a patient suffering from uterine cervical cancer who developed renal amyloidosis and rapidly progressive renal failure, associated with persistent wedge-shaped low-density lesions on renal sonography.

A 36-year-old woman who had been suffering from uterine cervical cancer was admitted because of long-lasting fever, bloody stool and nephrotic syndrome with impaired renal function on October 29, 1990. On admission, her blood pressure was 100/54 mm Hg and temperature 37.5 °C. There was pitting edema in the lower legs. The abdomen was slightly distended and tender. Laboratory data were as follows; urinary protein 1.5 g/day, hematocrit 16.4%, total protein 5.4 g/dl, albumin 1.9 g/dl, serum creatinine 6.59 mg/dl, blood urea nitrogen 99.0 g/dl, uric acid 16.5 mg/dl. Prednisolone (10 mg/day) and indomethacin which had been given for treatment of tumor fever were continued. The first sonography performed on the 2nd hospital day revealed slightly enlarged kidneys with 2 distinct wedge-shaped low-density areas in the right kidney and a probable one in the left kidney (fig. 1). Because contribution of indomethacin to formation of wedge-shaped lesions was suspected, indomethacin was discontinued and sulindac was given. The 2nd sonography performed on November 7 disclosed another distinct wedge lesion in both kidneys. Plain computed tomograms of the abdomen performed on November 14 revealed almost normal kidneys in density and configuration, and there were no tumor masses around renal vessels which might cause renal infarction or renal vein thrombosis. Melena increased in frequency and severity after hospitalization, and therefore, blood transfusion was required in a total amount of 15 units in 1 month. The 3rd sonography performed on November 27, when serum creatinine was 7.42 mg/dl, again revealed slightly enlarged kidneys with 1 distinct wedge lesion in the right kidney and with 1 distinct and 1 probable wedge lesion in the left kidney. There were no notches in the kidneys suggestive of cortical loss. The patient died of sepsis and uremia on December 6, 1990. Because autopsy was refused, necropsy of the right kidney guided by sonography was performed, and 5 pieces of specimen were obtained. Microscopic examination of the kidney specimen revealed renal amyloidosis. There was neither evidence of renal infarct nor tumor cell infiltrate.

Renal loss was not observed at the relevant sites 4 weeks after appearance of wedge-shaped lesions and furthermore, the number and the site of wedge-shaped lesions have changed with time during 4 weeks. There was no pathological evidence of renal infarct in the necropsy specimens. These findings excluded the possibility of renal infarct in this patient. We considered that renal hypoperfusion was apt to occur in association with hypovolemia due to nephrotic syndrome and melena and was furthermore aggravated by use of prednisolone and nonsteroidal antipyretic drugs, leading to wedge-shaped lesions. Our case also suggests that wedge-shaped lesions would not be constant but variable with time in number and/or their site.

Fig. 1. First Renal sonography: Two distinct wedge-shaped low-density lesions in the right kidney (left) and a probable one (black arrow) in the left kidney (right).

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