Interventional Therapy for Bilateral Acute Renal Artery Embolism Caused by Paroxysmal Atrial Fibrillation

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Clinically persistent atrial fibrillation (AF) occurring with systemic embolism is not uncommon. In contrast, the incidence of embolism due to paroxysmal AF is far lower than that of persistent AF.[1] Cerebral arteries are the most common sites of emboli caused by AF, followed by the mesenteric artery, splenic artery, lower extremity arteries, and renal arteries. However, the occurrence of concurrent bilateral renal artery emboli is extremely rare in paroxysmal AF patients. Within the scope of the information, we have collected, only three similar cases have been reported.[2-4]

A male patient, aged 82 years, was hospitalized due to a history of paroxysmal palpitation for two years and recent recurrence for two days. On admission, physical examination showed clear consciousness, clear breathing sounds in both lungs without dry or moist rales, a soft abdomen, no abdominal tenderness or rebound tenderness, no percussion pain in the renal region, and no edema in the lower extremities. The examination also revealed the following cardiovascular aspects: blood pressure (BP) of 140/60 mmHg, heart rate (HR) of 90 beats/min, definite arrhythmia, unequal intensity of the first heart sound, pulse deficit, and grade 2/6 systolic murmur in the mitral valve region. This patient had a previous history of arrhythmia, paroxysmal AF, and hypertension. Auxiliary examinations were performed after admission, as follows. Electrocardiogram (ECG) showed ectopic beats and AF. Doppler echocardiography showed an enlarged left heart, mild mitral insufficiency, and decreased left ventricular diastolic function. Laboratory test results were a serum creatinine (Scr) level of 79 μmol/L and blood urea nitrogen (BUN) level of 5.03 mmol/L. The diagnoses of the patient were (1) arrhythmia, AF, and heart failure with a New York Heart Association Functional Classification of IV; and (2) hypertension. The patient was administered rhythm-control therapy and anti-platelet therapy. Three days after admission, an ECG showed sinus rhythm and an HR of 49 beats/min. On the afternoon of that day, the patient had middle and lower abdominal pain (without radiating pain of the genitals), presenting as angina. He also had oliguria, urine with a dark yellow color, and nausea without vomiting. Physical examination revealed a soft abdomen, middle and lower abdominal tenderness but no rebound tenderness or muscular tension, percussion pain in bilateral renal regions, and bowel sounds of 3–5 times/min. Emergency abdominal Doppler ultrasound indicated no obvious abnormalities of liver, gall bladder, spleen, or kidneys. Emergency blood biochemistry tests showed a Scr level of 638 μmol/L and BUN level of 20.20 mmol/L, and acute renal failure was suspected. Renal computed tomography angiography (CTA) of the renal arteries showed filling defects in both proximal renal arteries (suspected emboli) [Figure 1a]. After consultation of doctors in the Department of vascular surgery, interventional therapy was suggested. Emergency arteriography of the renal arteries was performed bilaterally, and partial occlusion of both renal arteries was found [Figure 1b]. The decision to replace the 8F sheath was made. Then, the 8F guiding catheter (Beijing Life

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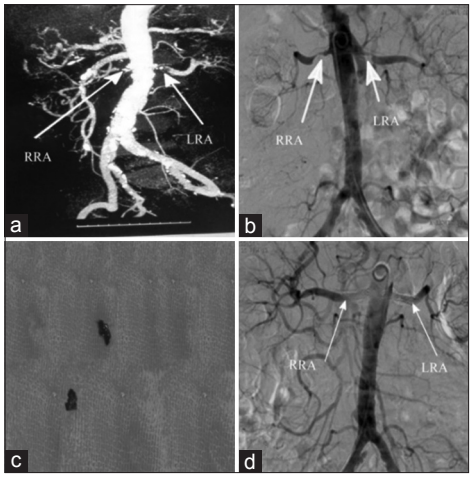

Figure 1: Imaging manifestations of preoperative, intraoperative and postoperative of intervention treatment on renal artery embolization (a) CTA of the renal arteries showed filling defects (as the arrows pointing) in both proximal renal arteries (suspected emboli). (b) Renal artery angiography showed bilateral renal artery root filling defect (as the arrows pointing), suggestive of emboli; (c) the thrombi were removed during surgery. (d) Normal blood flow in both renal arteries was restored after surgery (as the arrows pointing). RRA: Right renal artery; LRA: Left renal artery; CTA: Computed tomography angiography.

Oasis Technology Co., Ltd. Beijing, China) was inserted to remove the thrombi in both renal arteries. Several dark-red thrombi were identified [Figure 1c]. After that, 150,000 units of urokinase (Techpool Biochemical Pharmaceutical Company) were slowly injected into both renal arteries. Normal blood flow in both renal arteries was restored after surgery [Figure 1d]. Kidney function tests after surgery showed the following: (1) Scr levels of 279 μmol/L at 8 h, 152 μmol/L at 24 h, and 112 μmol/L at 3 days; and (2) BUN levels of 15.03 mmol/L at 8 h, 11.03 mmol/L at 24 h, and 7.03 mmol/L at 3 days. The patient was considered cured in one week and discharged.

In this patient, AF had already been present for over 48 h upon hospital admission. Three days after admission, the AF spontaneously disappeared, and the patient showed sinus rhythm. However, the patient soon presented with symptoms of persistent middle and lower abdominal pain, nausea, and oliguria, but without fever or chills. Physical examination revealed diffuse pain in the middle and lower abdomen combined with mild percussive pain in the region of each kidney. Kidney function test results were abnormal, so acute renal failure was suspected. In general, the causes of acute renal failure fall into three categories: prerenal, renal and postrenal. A common prerenal cause is a renal hypoperfusion (probably due to acute hemorrhage); other prerenal causes are heart failure and renal artery embolism. Renal causes include rapidly progressive glomerulonephritis, acute interstitial nephritis, and acute renal tubular necrosis. The most common postrenal cause is urolithiasis or tumor. Auxiliary examinations after admission excluded renal and postrenal causes. Acute hemorrhage and heart failure were also ruled out because the patient had normal BP and other vital signs, without the manifestations of acute heart failure. Considering that the AF disappeared spontaneously without anti-coagulation therapy, renal artery embolism was suspected and later confirmed by CTA. The common practice in treating renal artery embolism is to restore the blood supply to the kidneys through thrombolytic therapy, interventional therapy, and anti-coagulation therapy.[5] The patient described in this study received anti-coagulation therapy and achieved good restoration of blood flow to both kidneys.

The time from the onset of AF to the AF-related embolism was 5 days, after which the AF disappeared spontaneously, and the patient showed sinus rhythm. This finding implies that the risk of embolism caused by paroxysmal AF that lasts for over 48 h is very high. Taking this into consideration, risk stratification screening using the congestive heart failure, hypertension, age ≥75 years (doubled), diabetes, stroke (doubled), vascular disease, age 65–74 years, and sex category (female) (CHA2DS2-VASc) scoring system becomes very important.[6] Patients with a score of 2 or higher should receive anti-coagulation therapy with warfarin and other novel oral anticoagulants. The patient in this study was evaluated for embolism risk after hospital admission and had a score above 2. Although warfarin therapy was recommended to the patient and his relatives, this therapy was rejected due to fear of hemorrhage. The direct cause of bilateral renal artery embolism might be due to AF.

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Conflicts of interest

There are no conflicts of interest.

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