Successful treatment of acute renal infarction arising from left atrial myxoma and atrial fibrillation: A case report

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Abstract. Acute renal infarction (ARI) is an uncommon disease and delayed diagnosis or misdiagnosis often leads to rapid deterioration of the patient. As patients with atrial fibrillation and left atrial myxomas are at risk of developing ARI due to cardiogenic thromboembolism, it is pivotal for clinicians to make a correct diagnosis and start a timely treatment. The present study reported on the case of a 68-year-old man who suffered from ARI secondary to thromboembolism associated with atrial fibrillation and left atrial myxomas. His renal function deteriorated rapidly after the onset of the attack. He was treated with anti-coagulant agents with excellent results. Classical anti-coagulation therapy remains to be the effective and safe treatment of ARI.

Introduction

Acute renal infarction (ARI) is a severe condition requiring immediate treatment and is uncommon in clinical practice. It is often induced by the thrombogenesis or embolism to the renal artery trunk and branches, which leads to ischemic necrosis of renal tissues, kidney dysfunction and at times even irreversible renal parenchyma injury, leading to nephrectomy and even death in severe cases. As ARI does not have any typical clinical symptoms, it is easily misdiagnosed.

The majority of ARI cases included a case series have been reported to be of cardiac origin (1). A retrospective study on 438 ARI patients reported that cardiac factors were accountable for ARI in 55.7% of the cases (2), while atrial fibrillation and left atrial myxoma were common cardiac factors inducing systemic thromboembolism (3,4). Other risk factors include myocardial ischemia, coagulation disorders, hypertension, obesity, peripheral vascular disease, smoking, a prior thromboembolic event and diabetes (2,5). Anti-coagulant and anti-thrombotic agents are classical therapeutics for ARI. A previous study reported that percutaneous renal artery fibrinolytic therapy with re-vascularization, including aspiration and rheolytic thrombectomy, achieved an excellent result (6). However, operation trauma, post-operative complications and high mortality have limited the application of open surgery. Regarding the prognosis of renal infarction, a retrospective study of 67 patients with renal infarction reported that 41% patients suffered from acute kidney disease (AKD) (7). Another study with 20 months of follow up showed that 20% of ARI patients developed AKD, 11% developed new-onset renal insufficiency and 2.1% progressed to end-stage renal disease (2). Therefore, early diagnosis and timely treatment of ARI are pivotal for maintaining renal function and to improve the prognosis of patients affected. The present study reported on a patient with coronary artery disease (CAD), atrial fibrillation and left atrial myxoma, who developed ARI. His right kidney had lost its function due to atrophy and his renal function rapidly deteriorated to an uremic level over a period of 3 days.

The anti-coagulant agents low-molecular heparin and warfarin were used after timely diagnosis. His renal function eventually recovered and the renal infarction was effectively treated.

Case study

A 68-year-old male patient with a history of 20 years of coronary artery disease (CAD) and hypertension as well as 2 years of heart failure and atrial fibrillation was admitted to the Department of Cardiology, Changchun Central Hospital (Changchun, China) due to chest congestion and shortness of breath that had commenced 2 h previously. He had no diabetes and denied any history of kidney disease, which was in accordance with his medical records. Furthermore, the patient stated to have no drug or food allergies. Physical examination revealed that his blood pressure was 147/115 mmHg with a heart rate of 78 beats per minute. His body temperature was 36.8˚C and blood oxygen saturation was 98%. His breath sounds were clear with no rales, rhonchi or wheezes. However, the heart rhythm was irregular. There

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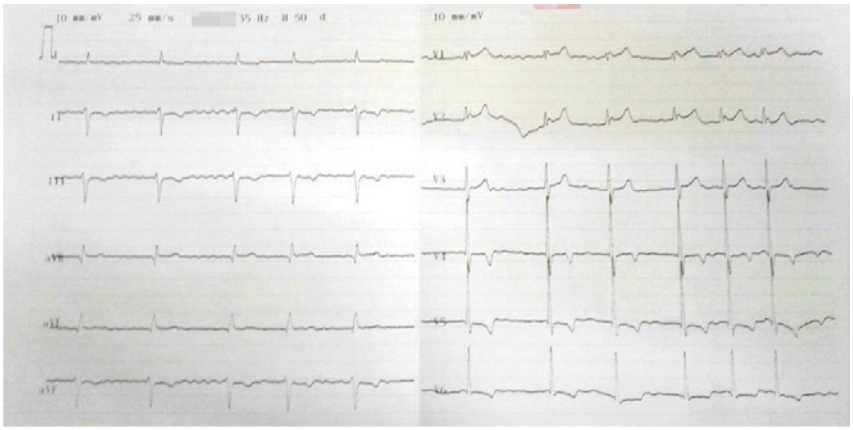

Figure 1. Electrocardiogram showing atrial fibrillation and left anterior parietal block.

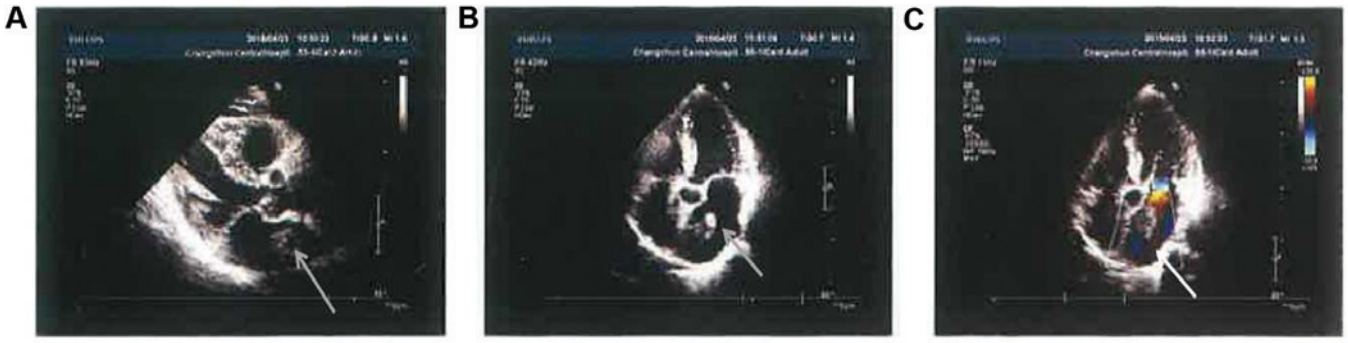

Figure 2. Cardiac ultrasound showing a 36x24.4 mm-sized echo in the left atrium, whose pedicle was bound to the left atrial wall (arrows) at the initial onset of ARI. Size, position, ultrasonic character and blood flow were observed in the (A) left ventricular spindle section, which exhibited a low echo indicating the possibility of a myxoma, confirmed when compared with (B) the four chambers of the heart. (C) A color Doppler ultrasound of the four chambers demonstrates the blood flow of the tumor itself.

was no cardiac murmur or pericardial friction sound. There was no edema in either of his lower extremities. Laboratory analysis showed that routinely assessed blood parameters, coagulation factors and myocardial enzymes were normal. His urine protein and urine occult blood culture results were both 3+. The red blood cell count in his urine was 188.4/μl. Serum nitrogen was 8.7 mmol/l (normal range, 2.8-7.6 mmol/l) and creatinine was 158.7 μmol/l (normal range, 35-132 μmol/l). His glomerular filtration rate was 39.8 ml/min (normal range, >60 ml/min). The electrocardiogram showed atrial fibrillation and left anterior fascicular block (Fig. 1). The renal ultrasound showed that the left kidney size and renal blood flow signals were normal. The right kidney size was 4.0x2.0 cm, the echo was uneven and the renal blood flow signals were reduced. The beginning segment of the right renal artery was normal but the middle segment was not clear, which implied right renal atrophy. Cardiac ultrasound showed left atrial myxoma, ventricular segmental wall motion abnormality and left ventricular diastolic dysfunction (Fig. 2). The diagnosis on admission was CAD, unstable angina pectoris, atrial fibrillation, heart failure class II, left atrial myxoma and hypertension. The patient initially refused anti-coagulation therapy.

The patient soon developed severe left-sided flank pain accompanied with nausea and oliguria of 400 ml per 24 h after the second day in hospital. There was no fever, chills, dysuria or hematuria. Aside from costovertebral angle tenderness, his physical exam was unremarkable and remained unchanged throughout the hospital stay. Creatinine was elevated to 744 μmol/l and the glomerular filtration rate was suddenly reduced to 8.3 ml/min. While lactate dehydrogenase (LDH) was normal on admission, it was elevated to 830.3 IU/l (normal range, 35-215 IU/l) at the second day of admission. The computed tomography (CT) examination of the abdomen showed right kidney atrophy and right kidney stones. As these factors were indicative of ARI, a contrast-enhanced CT scan of the abdomen was performed on day 2 after admission and renal infarction was confirmed (Fig. 3). Subsequently, the treatment was adjusted and anti-coagulant therapy was initiated, including low-molecular heparin (subcutaneous injection of 4,000 IU twice a day to bridge the warfarin treatment). Their dosage was adjusted until the coagulation routine international standardization ratio reached the target of 2-3. After systemic anti-coagulant therapy, the patient's abdominal pain alleviated gradually and the pain resolved on the 5th day. His urine volume recovered to normal. Urine analysis showed urine protein 1+ and no urine occult blood. His renal function improved after 1 week and creatinine was reduced to 191.6 μmol/l 1 month later (Fig. 4). The patient was kept on anti-coagulant therapy after 14 days, when the patient was discharged and their condition and renal function were stable at the follow-up exam on the 40th day.

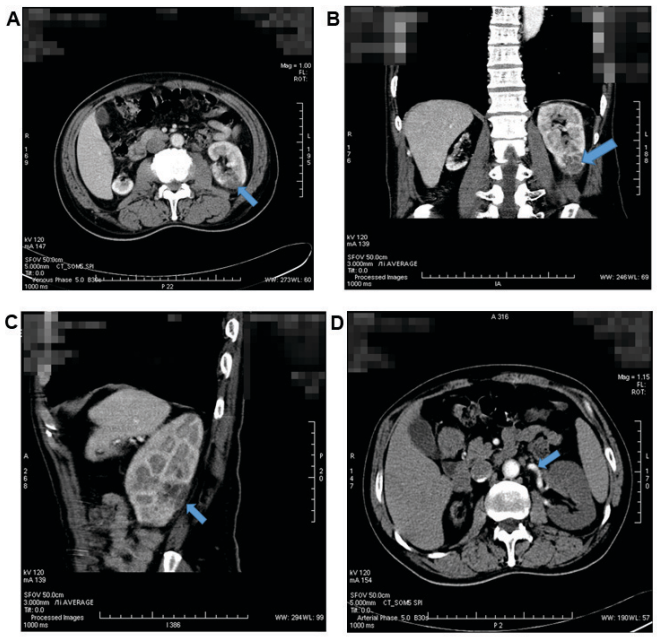

Figure 3. Contrast-enhanced computed tomography image with (A) axial, (B) coronal and parasagittal scanning, showing (C) multiple low-density perfusion changes among the left renal parenchyma and (D) severe stenosis of the left renal artery (arrows). Atrophy in the right kidney was also visualized.

Discussion

ARI is a rare condition with an incidence of 1.4% determined post-mortem and 0.007% in hospitalized patients (6). As its clinical manifestation lacks specificity and the specific indicators or biomarkers are currently unable to specifically identify the condition, it is frequently misdiagnosed or diagnosis is commonly delayed (8). Atrial fibrillation is the most important factor in the pathogenesis of ARI (3,6). When people suffer from atrial fibrillation, the endocardium is damaged, imbalances of thrombotic and thrombolytic mechanisms occurs, leading to the formation of an embolus, which may dislodge and travel through the systemic circulation, resulting in embolism in end organs. Furthermore, high expression of matrix metalloproteinases may incite degradation of the extracellular matrix and lead to the proliferation of myxoma cells into the vessel wall, inducing the occurrence of thrombosis (9). Atrial fibrillation and left atrial myxoma are risk factors for embolism. Early diagnosis is important to restore blood perfusion and renal function (10). The diagnosis and treatment in the present case proceeded as follows: The patient suffered sudden left abdominal pain. The CT exam showed right kidney atrophy and stones. While the most probable diagnosis was obstructive nephropathy, urine volume was obviously decreased and serum creatinine was sharply increased. If the left kidney had the compensatory capacity, his renal function would not have deteriorated so rapidly. Furthermore, the patient had previously suffered from coronary heart disease and had atrial fibrillation and left atrial myxoma as well, all of which are risk factors of thromboembolism. Furthermore, the patient did not consent to receiving anti-coagulant therapy before the occurrence of ARI and his LDH increased significantly, which suggested the possibility of renal infarction. Indeed, the contrast-enhanced CT confirmed this diagnosis. The left renal infarction caused a

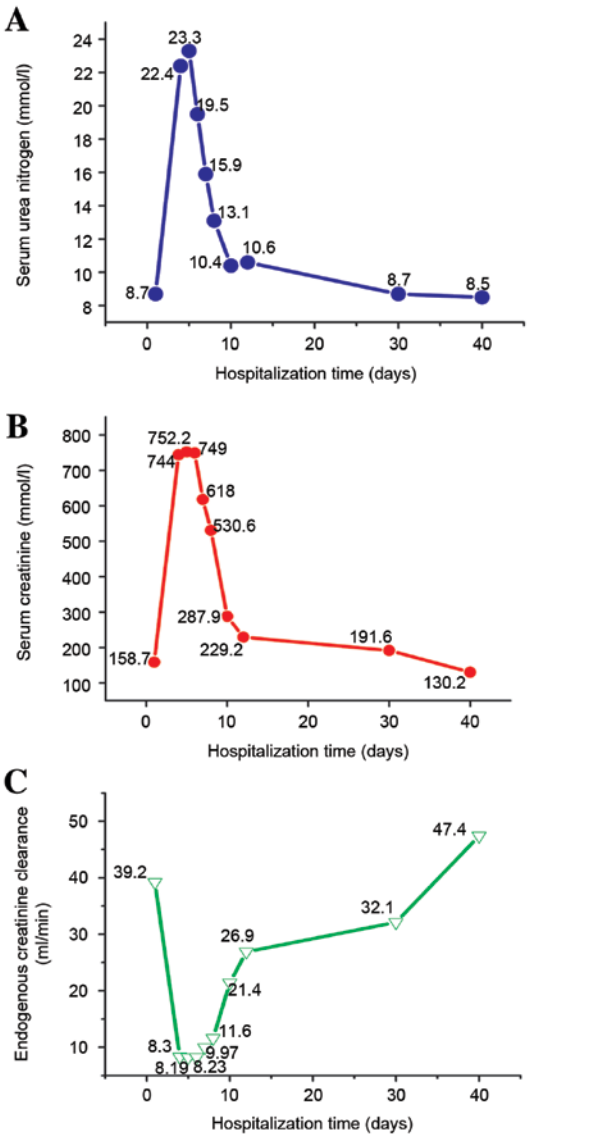

Figure 4. (A) Serum nitrogen, (B) serum creatinine and (C) glomerular filtration rate on different days after admission.

deterioration of his renal function, as his right kidney became atrophied and was not functional.

Anti-coagulation is a necessary and classical therapy for artery infarction and should be adopted as soon as possible to restore the renal perfusion and improve the prognosis (6,11). There are also cases of renal infarction that were successfully treated by percutaneous interventional artery thrombolysis on treatment of (6); however, this treatment is invasive. In view of the common trauma and complication of open surgical thrombectomy procedures and the poor tolerance of patients, it has rarely been used. The patient was an aged male and had an underlying disease accompanied with cardiac and renal insufficiency. His tolerance was poor and invasive treatment would have been associated with high risks. After careful contemplation considering the potential benefits and risks, anti-coagulation therapy was first selected. The left renal infarction led the deterioration of the function of the patient's right kidney, which had atrophied and was likely to have lost its function. After the systemic anti-coagulant treatment, the patient's renal function obviously recovered and the treatment

was effective, suggesting that anti-coagulation remains to be an effective approach for ARI. The patient did not undergo hemodialysis or any invasive operation but effectively retained his blood flow and renal function. The present case exemplifies that anti-coagulation treatment is worth considering as an effective treatment for ARI. Anti-coagulant agents have an irreplaceable role in the therapy of thromboembolism (12). Furthermore, warfarin has been shown to reduce the occurrence of systemic embolism (3). Compared to placebo-treated controls, warfarin reduced the risk of stroke by 60% in atrial fibrillation patients (13). A previous study also reported that warfarin significantly reduced the risk of myocardial infarction or stroke and was not correlated with the severity of chronic kidney disease (14). In the present case, to reduce risk factors of bleeding such as CAD, renal insufficiency and hypertension, coagulation parameters and renal function were comprehensively monitored, and the dosage of the agents was adjusted to minimize the risk of bleeding. Certain deficiencies must be acknowledged in the present case: Renal single-photon emission CT perfusion imaging should have been performed to evaluate the glomerular filtration of the bilateral kidneys, but the patient refused to take this test.

In conclusion, ARI is a fatal thrombotic disease. For old patients who suffer from heart disease and experience atrial fibrillation and left atrial myxoma, clinicians should pay close attention to factors such as urine volume and renal function. Furthermore, it is important for clinicians to adopt individualized solutions for specific cases of renal infarction to improve the prognosis. In the treatment of ARI, certain clinicians prefer invasive interventions and ignore the traditional anti-coagulant therapy. The present case suggested that anti-coagulation therapy remains an effective and pivotal treatment and has a definite therapeutic effect. Under the premise of strict consideration of indications and contraindications, it should not be neglected.

Acknowledgements

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