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Case Report

Renal arterial atherothrombosis due to catheter-induced dissection: Necessity of urgent intervention in a patient with a solitary functioning kidney

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ABSTRACT

Background: Renal artery stenosis (RAS) is a leading cause of hypertension, renal failure, pulmonary edema, and loss of renal mass. Atherothrombotic renal disease is a well-described entity, known primarily for its high mortality rate.
Case: Here, we present a case of acute unrecognized atherothrombosis of RAS due to catheter-induced dissection in a patient with a limited functioning renal artery and solitary kidney. A fifty-two-year-old patient recently diagnosed with hypertension was admitted to our cardiology clinic showing symptoms of uncontrolled hypertension. A renal angiography revealed 90% stenosis in the proximal segment of the right renal artery and totally occluded left renal artery. We decided to perform a percutaneous revascularization. However, the patient did not accept it. During follow-up, the patient's clinical condition deteriorated abruptly due to acute iatrogenic atherothrombosis. A percutaneous transluminal angioplasty was performed, and the patency of the renal artery was secured.
Conclusion: Renal angiography may cause acute iatrogenic atherothrombosis due to catheter-induced dissection in patients with solitary functioning kidneys. Percutaneous transluminal angioplasty may secure the patency of the renal artery.

Introduction

Renal artery stenosis (RAS) is an important cause of hypertension, renal failure, pulmonary edema, and loss of renal mass.[1, 2] The incidence of RAS varies from 5% to 29% according to previous angiographic studies.[1] About 90% of patients with RAS have atherosclerosis, 10% have fibromuscular dysplasia, and a few patients have arteritis or rare syndromes.[3] Although duplex ultrasonography, computed tomographic angiography, and magnetic resonance angiography are useful methods to detect RAS with high accuracy, catheter angiography is considered the gold standard for visualization of renal arteries.[3] Renal arterial revascularization is recommended in patients with resistant hypertension, deteriorating renal function, and single functioning kidney causing heart failure (flash pulmonary edema).[4] Although percutaneous intervention has proven beneficial, there are no strict guidelines for timing courses of treatment according to clinical features of atheromatous RAS.[5, 6] In this report, we present a case of acute unrecognized atherothrombosis of RAS due to catheter-induced dissection in a patient with a limited functioning renal artery and solitary kidney.

Case report

A fifty-two-year-old patient recently diagnosed with hypertension was admitted to our cardiology clinic showing symptoms of uncontrolled hypertension. Upon his diagnosis, he was prescribed a treatment of carvedilol, amlodipine, valsartan/hydrochlorothiazide, and doxazosin. On physical examination, the patient's arterial blood pressure was 175/100 mm/Hg. A systolic murmur was heard on the periumbilical area of the abdomen. Upon laboratory analysis, his creatinine and urea were 1.49mg/dl and 54 mg/dl, respectively. Creatinine clearance was calculated as 58 ml/min by the Cockcroft-Gault equation. Renal artery Doppler ultrasonography revealed that the right renal arcuate artery resistive index (RI) was observed to be high, which was 78. No blood flow was observed in the left renal artery. Renal ultrasonography revealed an atrophic left kidney, in which cortico-medullary differentiation was not possible. An abdominal and renal angiography performed with nearly 25 cc contrast medium demonstrated 90% stenosis in the proximal segment of the right renal artery and total occlusion of the left renal artery (**video 1**). Although

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Fig. 1. After the placing an 8 × 15 mm renal stent (Abbott Vascular, USA) through the right renal artery.

we decided to proceed with a percutaneous revascularization, the patient refused this course of treatment. Four hours after the procedure, the patient's clinical condition deteriorated abruptly. He developed flush pulmonary edema and uncontrolled hypertension. Intravenous nitrate and diuretic began to stabilize his clinical condition, but then the patient regressed and exhibited symptoms of nausea, vomiting, and mild pain in the right upper quadrant and abdomen. He became anuric within a few hours. An urgent hemodialysis was performed, and it was decided to revascularize the right renal artery twelve hours after renal angiography. Unlike the first angiography, the second angiography showed total occlusion of the right renal artery, most possibly due to an overlooked catheter-induced dissection (**Video 2**). An 8 × 15 mm renal stent (Abbott Vascular, USA) was placed through the right renal artery, and total patency of the right renal artery was secured (Fig. 1). Forty-eight hours after the renal revascularization, the patient showed signs of diuresis. He underwent hemodialysis four times during the post-intervention period. The patient was well in the follow-up and did not need hemodialysis after hospital discharge. His renal functions improved and returned to baseline urea and creatinine values. According to an additional follow-up, his creatinine was 1.42 mg/dl and urea was 51 mg/dl after one year.

Discussion

Critical RAS was defined as a reduction of more than 60% in renal artery diameter.[7] Renal perfusion reduced by RAS may lead to hypertension, renal dysfunction, loss of renal mass, and/or pulmonary edema. Despite improvements in diagnostic and interventional techniques, controversy remains over whether and how to revascularize the kidneys of patients with stable RAS. Moreover, there is no definite timing of revascularization.[5, 6] Because of RAS of solitary functioning kidney (right renal artery) and uncontrolled hypertension, we decided to revascularize renal artery, but the patient refused to undergo the procedure. After the first renal angiography, our patient's clinical condition rapidly deteriorated. After observing a total occlusion of the right renal artery in the second angiography, we deduced it was an acute iatrogenic atherothrombosis caused by catheter-induced dissection, since we observed no risk factors for peripheral arterial emboli such as valvular heart disease, prosthetic heart valves, dilated cardiomyopathy, and arrhythmias.[8] With a 38% rate of mortality,[9] atherothrombotic renal disease can occur spontaneously or as a result of atherosclerotic debris being released from surgical manipulation, vascular interventions with angiographic catheters.[10] Platelet degranulation, pro-coagulant molecules, and platelet-derived growth factors might contribute to acute thrombosis and the rapid total occlusion of the renal artery. Renal salvage with acute renal occlusion has a short window to restore blood flow and preserve renal function. Some studies showed that ischemic renal parenchymal injury occurred in as short a period as 1–3 h.[11, 12] When a patient presents early with acute renal infarct, three management options are available: surgical treatment, percutaneous transluminal intervention (PTI), and thrombolysis. Thrombolytic therapy may be associated with incomplete main renal artery patency, or with obstruction of more distal segmental branches. Furthermore, if ischemia has persisted for more than 90 minutes, thrombolytic therapy does not restore renal function.[12] Hence, thrombolytic therapy was not a suitable option for our case. When performing PTI, the results are variable without stent placement due to the risk of distal embolization, which may cause complete obstruction distally in a smaller branch. As shown in our case, stent placement can reduce distal embolization and provide full patency in a total occluded vessel.

Conclusion

Based on our observations, renal angiography may cause acute iatrogenic atherothrombosis due to catheter-induced dissection in patients with solitary functioning kidneys. Percutaneous transluminal angioplasty may secure the patency of the renal artery.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to authorship, and/or publication of this article.

Video 1: Abdominal aortography and renal angiography showed 90% stenosis of proximal segment of the right renal-artery and the total occlusion of left renal artery.

Video 2: Second renal angiography showed the total occlusion of the right renal artery.

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Supplementary data

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