Case Report

Spontaneous renal artery thrombosis: A rare cause of acute flank pain

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Spontaneous renal artery thrombosis is a rare cause of flank pain and can have fatal consequences. We report a case of acute renal artery thrombosis in a 61-year-old man who experienced flank pain and had no medical history. A contrast-enhanced computed tomography scan revealed total thrombotic occlusion of the left renal artery. The patient was taken to interventional radiology, and an urgent catheter-directed thrombolysis of the renal artery was performed. The procedure was successful, with the subsequent arteriogram demonstrating a substantial decrease of the thrombus extent and the recanalization of the left renal artery. This case highlights that emergency renal artery thrombolysis is an effective and safe treatment for acute occlusion of the renal artery.

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Introduction

Renal artery thrombosis is a rare cause of acute flank pain and requires immediate medical intervention. Previous thromboembolic events, cardiovascular disease, chronic atrial fibrillation, trauma, and aortic interventions are common risk factors [1–3]. Spontaneous renal artery thrombosis is extremely rare, and few reports have been documented to date [3,4]. The clinical implications of acute renal artery thrombosis vary from severe abdominal pain or abrupt flank pain to both symptoms [3]. Thus, prompt diagnosis upon the appearance of flank pain should be performed before irreversible renal damage occurs. We report the case of a 61-year-old man with spontaneous left renal artery total thrombotic occlusion, which after catheter-directed thrombolysis (CDT) led to partial recanalization of the renal artery and complete symptom resolution.

Case report

A 61-year-old man presented left flank pain the day before seeking medical attention. He had no known medical history and thus no history of abdominal aortic aneurysm or a

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previous vascular intervention. A physical examination provided the following vital signs: body temperature of 36.6°C, blood pressure of 174/115 mmHg, heart rate of 81 beats per minute, respiratory rate of 16 breaths per minute, and oxygen saturation of 99% on room air acquired from pulse oximetry. No significant findings were found on the results of complete blood count, blood chemistry, electrolytes, and coagulation profile including the D-dimer test, except for an increase in lactate dehydrogenase (LDH) to 1087 U/L and a serum blood urea nitrogen/creatinine level of 13.3/1.39 mg/dL. In addition, the echocardiogram showed a normal sinus rhythm. The patient underwent a contrast-enhanced computed tomography examination for further evaluation. The scan revealed near complete infarction of the left kidney and thrombotic occlusion at the left proximal renal artery without underlying anatomic abnormality of the renal artery (Fig. 1).

We decided to use thrombolitics through percutaneous intervention given that no contraindication existed for this treatment. Through the right femoral artery, a renal artery angiogram was obtained. It showed near-total thrombotic occlusion of the left proximal renal artery and nonvisible distal flow. The occlusion segment of the left renal artery was cannulated with a 1.7F microcatheter (Progreat Lambda; Terumo, Somerset, NJ, USA) and a 0.016-inch microwire (Meister; Asahi Intecc, Nagoya, Japan) (Fig. 2). Once the occluded area was traversed by the microwire, selective injections showed no dissection of the intrarenal branches. Thrombolysis was performed by placing a 7 cm multisideport catheter infusion set (Cook Medical, Bloomington, IN, USA) into the thrombosed left renal artery. First, urokinase was infused at 40,000 IU/h, and the patient was placed on intravenous heparin followed by oral warfarin. No adverse effects occurred, and abdominal pain disappeared immediately. Arteriography was performed after 13.5 hours to evaluate the response of thrombolysis and correct the catheter position. Infusion continued for additional 18.5 hours for resolution of the remaining thrombus burden. The subsequent arteriogram demonstrated a substantially lower thrombus extent and partial recanalization of the left renal artery (Fig. 3). Further medical evaluation was performed aiming to identify the hyperviscosity syndrome. The normal levels of protein C, protein S, and antithrombin III allowed to discard the most common primary hypercoagulable states. The
renal artery, we decided to perform CDT considering the patient's condition of neither active bleeding diathesis nor recent operation history.

Urokinase and recombinant tissue plasminogen activator are commonly used as thrombolytic agents in clinical practice. Various techniques are available for infusion of the thrombolytic agent. Specifically, the McNamara protocol combines high- and low-dose infusion, whereas the Hess technique requires manually injecting a small bolus of 1-3 mL of urokinase (4000 U/mL), and the Bookstein pulsed-spray technique consists of the intermittent injection of high-pressure spray [11–13]. We used the renowned McNamara protocol with graded tapering of infusion rate under restoration of antegrade flow. Systemic heparin infusion continued throughout CDT. Neither significant bleeding complications nor distal embolization were observed in the subsequent angiogram.

The duration of anuria is one of the most important prognostic factors after intra-arterial fibrinolytic therapy for renal thromboembolism [9]. Persistent anuria suggests poor prognosis and requires further surgical management. No anuria was observed in the reported case, assuring recovery of renal function after restoration of blood flow. However, in previous studies, patients with complete occlusion of the main renal artery persisting over 3 hours who underwent surgical or fibrinolytic intervention presented irreversible damage. Moreover, renal parenchyma and renal function were not recovered after restoration of renal blood supply regardless of the therapeutic strategy [14]. Therefore, routine follow-up should be clinically recommended after treatment.

We should suspect spontaneous renal artery thrombosis as a differential diagnosis in the setting of renal colic and elevated LDH levels. A high suspicion of this diagnosis can be conductive to early treatment and organ salvage.

Discussion

Acute renal artery thrombosis is a rare case that requires fast diagnosis and prompt treatment to preserve renal function. Most patients suffer from a sudden, sharp, unremitting pain in the flank or upper abdomen/lower back pain. This may be accompanied by fever, nausea, vomiting, and leukocytosis [5]. The disease has been characterized in previous studies, and it is usually associated with other causes such as trauma, vasculitis, instrumentation, sepsis, transplant, sickle cell disease, and antiphospholipid syndrome [6]. Spontaneous renal artery thrombosis in an otherwise normal renal artery is very rare, and few cases have been reported to date [7].

The reported case was characterized by left flank pain, elevated LDH levels, and thrombus in the left proximal renal artery. Like in [4], serum LDH levels and contrast-enhanced computed tomography contributed to diagnosis. We considered renal infarction in an elevated serum LDH with small or no increase in serum aminotransferases [8]. The radiologic features were also consistent with renal infarction due to partial thrombus. Given the rarity of this disease, the treatment guideline was not reached on the agreement. As a minimally invasive technique, local percutaneous intra-arterial fibrinolytic therapy can be applied to recanalize acute renal artery occlusions of variable duration ranging from less than 24 hours to several weeks and provide a convenient alternative to surgical intervention in selected cases [9]. Therefore, angiography is still considered as the gold standard to confirm the diagnosis of acute renal artery thrombosis [3,10]. In the reported case of near-total thrombotic occlusion of the main

Authors’ contributions

LHJ: Acquisition and analysis of the work, Drafted the work. HJJ: Conception of the work and substantively revised it. KJH: Design of the work and substantively revised it. LKH: Writing – review & editing. PS: Writing – review & editing. PSH: Writing – review & editing. All authors have checked the authorship to a submitted version and agreed to the author list and contributions.

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