CASE REPORT

Renal artery pseudoaneurysm presenting 3 years after deceleration injury

Steven Weissbart, BS, Justin Han, MD, Ojas Shah, MD
Department of Urology, Bellevue Hospital, New York University School of Medicine, New York, New York, USA

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Renal artery pseudoaneurysms (RAPs) are vascular lesions that have most commonly been reported secondary to penetrating renal trauma and iatrogenic injury. We present the first case of a RAP that developed as a result of an isolated deceleration injury. The patient presented 3 years after his injury with symptoms of gross hematuria, right flank pain, and syncope. Diagnosis of his RAP was made by magnetic resonance imaging (MRI) and angiography. He was successfully treated with angioembolization.

Key Words: aneurysm, embolization, false, trauma, renal, hematuria

Introduction

Renal artery pseudoaneurysms (RAPs) develop when trauma to an arterial wall leads to the development of an extravascular hematoma that freely communicates with the intravascular space. These vascular lesions have most commonly been reported secondary to penetrating renal trauma and iatrogenic injury (e.g. partial nephrectomy and percutaneous renal procedures).

The most common presenting symptoms of RAPs include gross hematuria, flank pain, abdominal mass, and hypertension. The length of time from the inciting traumatic event until presentation is typically days to weeks.

We describe a case of a renal artery pseudoaneurysm that uniquely developed secondary to a deceleration injury and spontaneously presented with life threatening gross hematuria 3 years after the patient's reported trauma.

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Address correspondence to Dr. Ojas Shah, Chief of Urology, Bellevue Hospital, NYU School of Medicine, 150 E. 32nd Street, 2nd Floor, New York, NY 10016 USA

Case report

A 44-year-old man presented to the emergency room with the acute onset of gross hematuria, right flank pain, and syncope. His past medical history was significant only for a rock climbing accident 3 years prior to presentation. While climbing a gymnasium rock climbing wall, the patient slipped and in freefall grabbed onto a protruding boulder, holding it with his right hand in order to prevent his fall. The patient reported that he had no contact between his body and the wall; however he was wearing rock climbing gear, including a harness, and was suspended with rope. In the ensuing hours he developed ecchymosis extending from his right axilla to flank. He denied any significant flank pain, gross hematuria or shortness of breath at that time. The patient never sought medical attention at that time.

In the emergency room, his vital signs were stable. His abdomen was soft, nontender and nondistended with no palpable masses. He had no costovertebral angle tenderness. Laboratory results returned a hematocrit of 19.9 and a serum creatinine level of 1.3. A Foley catheter was placed, revealing moderate blood with clot. CBI was started and his hematuria slowly cleared.

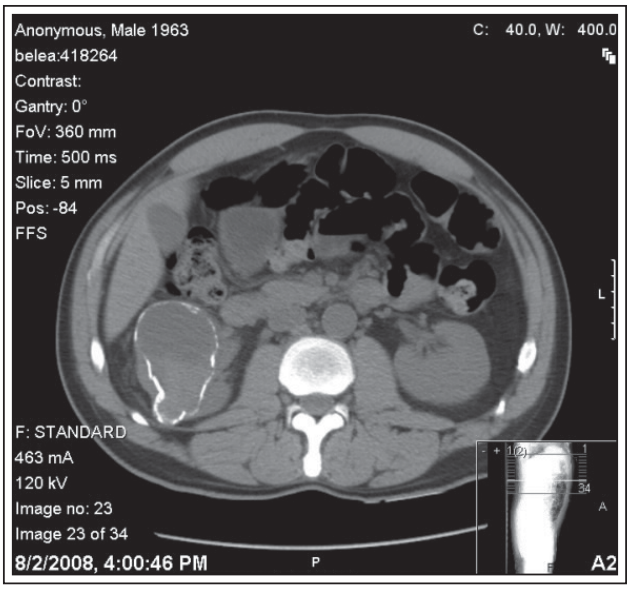

Figure 1. Axial computed tomography scan (CT) demonstrating cystic lesion in right kidney with dense irregular calcifications suggestive of a hemorrhagic cyst.

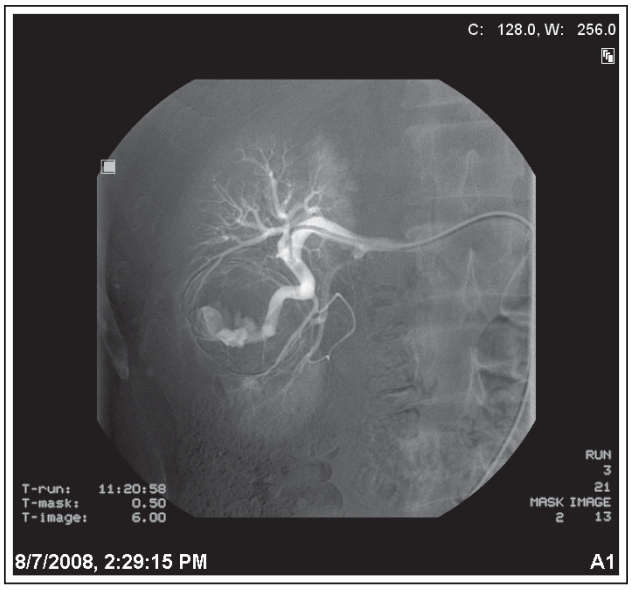

Figure 3. Selective renal artery angiogram demonstrating a hypertrophied branch of right renal artery with gross extravasation of contrast, establishing the diagnosis of a renal artery pseudoaneurysm.

The patient was transfused six units of packed RBCs and a computed tomography scan (CT) scan was obtained. Imaging revealed a calcified cystic lesion in the right kidney with intralesional hemorrhage, consistent with a Bosniak III complex renal cyst, Figure 1. To further evaluate this lesion, a follow up MRI demonstrated a hypertrophied right renal artery branch leading to the centrally located complex cystic renal lesion, raising suspicion of a renal artery pseudoaneurysm, Figure 2.

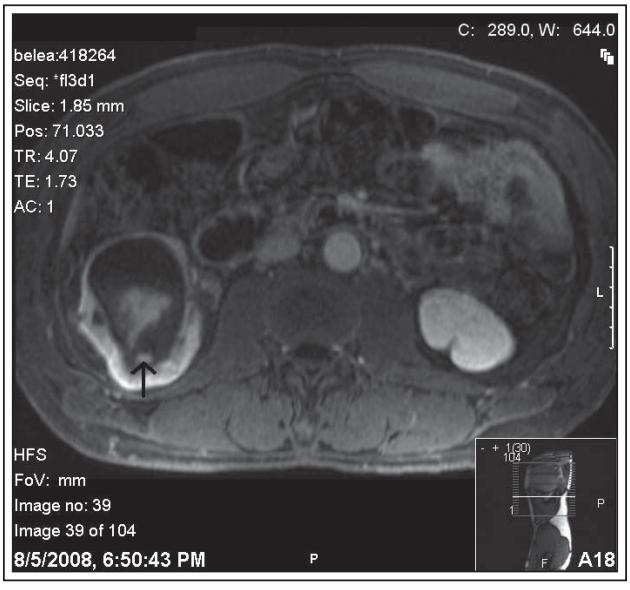

Figure 2. Axial magnetic resonance image (MRI) demonstrating cystic lesion in right kidney with focal 9 mm nodule of enhancement (arrow). This area of enhancement is the cross section of a hypertrophied branch of right renal artery supplying the region suggestive of a renal artery pseudoaneurysm (T1 weighted, post contrast subtracted image).

Selective right renal angiography revealed gross extravasation of contrast from a hypertrophied branch of the right renal artery into a large intrarenal pseudoaneurysm, Figure 3. Transcatheter coil embolization was then performed using three micro Nestor coils. Postembolization films showed complete cessation of blood flow into the pseudoaneurysm.

The patient was discharged 3 days after embolization and is doing well 4 months later.

Discussion

Renal artery pseudoaneurysms form following trauma to a branch of the renal artery. A subsequent combination of coagulation, hypotension, and compression of the artery by the surrounding tissues (i.e. Gerota's fascia, renal parenchyma, and the vascular adventitia) temporarily stops the bleeding. Over time, the degradation of the clot and the surrounding necrotic tissue allow the extravascular space to recanalize with the intravascular space, leading to the development of a pseudoaneurysm. With a return

to normal blood pressure, the pseudoaneurysm can dilate and potentially rupture into the perirenal space or the renal collecting system.

Renal artery pseudoaneurysms have most commonly been reported secondary to penetrating renal trauma, percutaneous renal procedures, and partial nephrectomy.[1-3] While blunt trauma is recognized as an etiology of RAPs, there are only about 20 reports of this in the literature. In these cases, the two potential mechanisms of RAP development include direct arterial injury caused by blunt force and laceration of the arterial wall due to the deceleration force that is associated with the blunt injury. The latter theory is supported by the observation that most patients who developed RAPs after blunt trauma were victims of motor vehicle accidents or falls from significant heights.

We present a case of a RAP that developed from a deceleration injury with no penetration injury and no documented direct blunt trauma to the flank. While deceleration forces may have led to the development of RAPs in patients who had suffered blunt trauma, it is impossible to elucidate whether the RAPs were truly caused by the deceleration force or the traumatic blow to the kidney causing direct arterial injury or some combination of the two forces. Given that our patient experienced a strong deceleration force isolated from any direct blunt trauma, this case substantiates the belief that deceleration forces alone may be responsible for RAP formation in some patients.

Not only is the etiology of this patient's injury of interest, but also noteworthy is the 3 year long time delay from injury until presentation. RAPs typically present within days to weeks after injury. In Heyns and Vollenhoven's series of six patients who developed a RAP from penetrating trauma, the mean time to presentation was 11.5 days (range, 4-36 days), excluding one patient who presented with microscopic hematuria at 18 months.[4] In Singh and Gill's series of six patients who were diagnosed with RAPs after laparoscopic partial nephrectomy, the mean time to presentation was on postoperative day 12 (range, 8-15 days).[3] Interestingly, the time to presentation in Lee and Porter's blunt trauma review is more variable (ranging from 9 days to 15 years).[5] This phenomenon can be explained by the fact that patients who endure penetrating trauma or undergo partial nephrectomy are more closely monitored than patients who suffer from blunt trauma. Additionally, as in our case, patients who suffer nonpenetrating trauma may not seek the same degree of medical attention as patients who have suffered penetrating injuries. Another explanation for the shorter time to presentation in patients with RAPs caused by penetrating injury and partial nephrectomy is that the integrity of these patients' urinary collecting systems may have been disturbed, thus facilitating the production of hematuria caused by the RAP.

Also of interest are the imaging modalities that were needed to diagnose the RAP in this case. While the calcified wall of the lesion seen in Figure 1 was detected on CT, MRI and angiography were required to make the diagnosis of a RAP. This is noteworthy because CT has previously been reported to be an effective modality to diagnose RAPs.[6] Aside from CT, other imaging modalities have also been reported to be effective in diagnosing RAPs. Sullivan et al demonstrated color Doppler sonography to be a useful diagnostic modality in their report of two patients with RAPs.[7] In this modality, RAPs are diagnosed by detecting a lesion with bidirectional swirling blood flow. Renal perfusion imaging studies have been shown to be useful in diagnosing RAPs as well. In their report of one patient with a RAP, Aburano et al utilized the Tc-99m DTPA study to demonstrate that RAPs have a localized increase of activity on blood flow image, which disappears on subsequent renal images.[8] Angiography is accepted as the gold standard modality in evaluating traumatic renal vascular lesions.[1] This modality is especially useful in that therapeutic embolization may be immediately conducted if a RAP is diagnosed.

RAPs are rare complications of renal trauma. These vascular lesions should be kept in the differential diagnosis of patients presenting with persistent hematuria, regardless of injury mechanism or time of trauma. □

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