

Interventional Radiology

Page kidney secondary to subcapsular hematoma following percutaneous renal allograft biopsy

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ABSTRACT

Percutaneous renal biopsy with ultrasound guidance is a helpful procedure regularly performed to obtain renal tissue diagnosis for rejection in the postrenal transplant setting; however, it is not without risks. We report the case of a 42-year-old male with end stage renal disease who developed a subcapsular hematoma, with subsequent hypertension and renal failure, compatible with acute page kidney as a complication of the renal biopsy. The ultrasound images demonstrated classic imaging appearances which all diagnostic and interventional radiologists should be aware of. The patient was managed successfully with conventional open surgical evacuation of the hematoma with return to baseline laboratories and vital signs after the procedure.

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Case report

A 42-year-old male with end stage renal disease secondary to chronic hypertension had a cadaveric renal transplant 5 months before the current presentation. As part of the posttransplant follow-up, he was noted to have a rising creatinine of 2.2 mg/dL from baseline of 0.6 mg/dL (NR: 0.5–1.4 mg/dL). He was referred for a percutaneous renal allograft biopsy. The biopsy was uneventful with adequate cores obtained to the satisfaction of the pathologist. The patient then returned a week later with rapid elevation of creatinine (14.9 mg/dL) and new onset hypertension (161/96 mm Hg, post-transplant baseline was 144/70 mm Hg). The patient was admitted and a stat ultrasonography (US) scan of the transplant kidney was performed.

Prebiopsy ultrasound with Doppler images demonstrated an unremarkable allograft kidney, without hydronephrosis. The main renal artery peak systolic velocity was 65.3 cm/s, and the renal artery (RA):Aorta velocity ratio was 0.5. The arcuate artery

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Fig. 1 - Forty-two-year-old male status after allograft kidney transplant with clinical pattern suspicious of rejection. Prebiopsy diagnostic ultrasonography scan demonstrates normal superior internal resistive indices. FINDINGS: Longitudinal color Doppler of the superior arcuate arteries of the transplanted kidney demonstrates a normal resistive index of 0.68. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.

resistive indices in the inferior, mid, and superior arteries were 0.68, 0.70, and 0.69, respectively (Figs. 1-3).

needle placed just outside the renal cortex. The tract was sealed with gelfoam, and no immediate complications were observed (Fig. 4).

Images from the percutaneous renal biopsy demonstrated an 18-gauge Biopence needle within the renal cortical paren-



Fig. 2 - Forty-two-year-old male status after allograft kidney transplant with clinical pattern suspicious of rejection. Prebiopsy diagnostic ultrasonography scan demonstrates normal mid internal resistive indices. FINDINGS: Longitudinal color Doppler of the mid arcuate arteries of the transplanted kidney demonstrates a normal resistive index of 0.70. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.

chyma. The needle was inserted through a 17-gauge coaxial



Fig. 3 – Forty-two-year-old male status after allograft kidney transplant with clinical pattern suspicious of rejection. Prebiopsy diagnostic ultrasonography scan demonstrates normal inferior internal resistive indices. FINDINGS: Longitudinal color Doppler of the inferior arcuate arteries of the transplanted kidney demonstrates a normal resistive index of 0.69. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.

The stat ultrasound with Doppler of the allograft kidney upon return to the emergency department demonstrated an $8.6 \times 3.0 \times 6.8$ cm heterogeneous, ellipsoid collection, compatible with a subcapsular hematoma. No hydronephrosis was seen (Fig. 5). The main renal artery peak systolic velocity had increased to 143 cm/s from prebiopsy velocity of 65.3 cm/ s. The RA:Aorta ratio had also increased to 0.7 from 0.5. The arcuate artery resistive indices in the inferior, mid, and



Fig. 4 – Forty-two-year-old male status after allograft transplant kidney biopsy presenting with acutely deteriorating renal function and acute hypertension. Ultrasonography scan image obtained during biopsy. FINDINGS: Longitudinal grayscale ultrasound of the right lower quadrant with an 18-gauge Biopence needle within the renal cortex. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.



Fig. 5 – Forty-two-year-old male status after allograft transplant kidney biopsy presenting with acutely deteriorating renal function and acute hypertension. STAT ultrasonography scan. FINDINGS: Longitudinal grayscale ultrasound of the right lower quadrant demonstrating an ellipsoid, heterogeneous mass indenting the renal cortex. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.

superior arcuate arteries had increased to 0.88, 0.86, and 0.82 from 0.68, 0.70, and 0.69, respectively. There was reversal of diastolic flow in the arcuate arteries (Fig. 6-8).

A dialysis catheter was placed, and the patient received emergent hemodialysis on the day of admission. The patient underwent emergency surgical evacuation of the subcapsular hematoma, the kidney was found to be still viable. After procedure the renal function returned to baseline function and the patient was discharged after 4 days.



Fig. 6 – Forty-two-year-old male status after allograft transplant kidney biopsy presenting with acutely deteriorating renal function and acute hypertension. STAT ultrasonography scan with Doppler evaluation. FINDINGS: Longitudinal color Doppler of the superior arcuate arteries of the transplanted kidney demonstrates an increased resistive index of 0.82. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.



Fig. 7 – Forty-two-year-old male status after allograft transplant kidney biopsy presenting with acutely deteriorating renal function and acute hypertension. STAT ultrasonography scan with Doppler evaluation. FINDINGS: Longitudinal color Doppler of the mid arcuate arteries of the transplanted kidney demonstrates an increased resistive index of 0.88. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.



Fig. 8 – Forty-two-year-old male status after allograft transplant kidney biopsy presenting with acutely deteriorating renal function and acute hypertension. STAT US scan with Doppler evaluation. FINDINGS: Longitudinal color Doppler of the inferior arcuate arteries of the transplanted kidney demonstrates an increased resistive index of 0.88. TECHNIQUE: Longitudinal image obtained with a 5 Mhz curvilinear probe.

Discussion

Hypertension due to compression of the renal parenchyma is a well-known phenomenon that was first observed and described in 1939 by Dr. Irvine Page when he wrapped a canine kidney in cellophane, thereby causing inflammation and constrictive perinephritis [1]. Years later, Engel and Page published a case of a young football player with hypertension due to a perinephric, subcapsular hematoma, which resolved with unilateral nephrectomy [2,3]. Subsequent research into this phenomenon has revealed that hypertension occurs due to microvascular ischemia, which results in activation of the reninangiotensin-aldosterone system [4,5]. Since the original description in 1939, page kidney has been observed in a number of patients, from a variety of causes including trauma, renal biopsy, cyst rupture, spontaneous hemorrhage, polyarteritis nodosa, and urinoma [6–10]. A comprehensive literature review of all published page kidney cases from 1955 through 2008 demonstrated that two-thirds of the patients were men, and the average age was 38 years (range, 17 to 69 years) [6,9].

Bleeding after renal biopsy is not an uncommon complication. In a prospective analysis of 471 patients, there was a reported 33.3% rate of postbiopsy hematomas, but 90.4% of the hematomas were clinically silent [2]. In another prospective study of 147 patients, 4 (5.6%) of 71 biopsied transplanted kidneys were noted to be complicated by hematoma when reevaluated the following day; however, it is not clear if these were subcapsular [11]. Schwaighofer et al. noted subcapsular hematomas in 17 (8.1%) of 98 ultrasound-guided biopsied transplant kidneys [12]. The incidence of patients with postbiopsy subcapsular hematomas that go on to develop page kidney is not known. A number of isolated page kidney cases after transplant kidney biopsies have been reported in literature [13–18]. A large retrospective study of 518 renal transplant biopsies, noted only 4 patients (0.8%) to develop biopsy-induced subcapsular hematoma resulting in page kidney [15]. Furthermore, Wanic-Kossowska et al. report just 3 cases (0.3%) of page kidney of 800 percutaneous renal biopsies performed over 10 years [19].

Page kidney usually presents with acute pain, oliguria, and hypertension in the background of a recent cause for subcapsular space occupying lesion [15]. In some cases the patient develops a significant decline in renal function, as in this patient. This is due to hypoperfusion of the kidney, hence decreased renal filtration ability.

Subcapsular hematomas can be imaged with computed tomography (CT) and magnetic resonance imaging (MRI), but only Doppler ultrasound can demonstrate the hemodynamic changes caused by the renal compression. Contrast enhanced CT will demonstrate a round or ellipsoid, high attenuation fluid collection indenting or flattening the renal margin. High attenuation fluid (40-70 HU) is consistent with acute clotted blood [20]. MRI findings are more varied depending on the nature of the subcapsular space occupying lesions. If blood is present, the MRI appearances would vary with the age of the hematoma. Acute blood (hours to 3 days) will demonstrate decreased T1 and T2 signal [21,22].

Similarly, sonographic features vary with time. Acute hematomas generally present as heterogeneously echogenic material and as the hematocrit effect of the hematoma progresses they may become more anechoic or cystic, with lowlevel echoes with or without septations. Doppler is particularly helpful in the diagnosis of page kidney, given the hemodynamic changes caused by the compression of the renal vasculature. Significant compression will cause increased resistance to flow, which depending on the severity has variable velocity changes ranging from increased peak systolic velocities to reduced velocities and not infrequently, in severe cases—loss of flow or reversal of the diastolic waveform. Resistive indices are also elevated [14,15,23,24].

Early recognition of acute page kidney is important to prevent progressive pressure induced ischemic organ damage. However, aggressive and prompt intervention to preserve as much renal function as possible is the most sensible approach. There is currently no evidence-based guideline for the management of page kidney. Initial attempts should be made to stabilize the patients conservatively with antihypertensive medication such as angiotensin converting enzyme inhibitors and strict fluid balance [25]. However, given the delicacies surrounding transplant kidneys, there is a relatively low threshold for operative intervention to preserve the transplant kidney. Although small, asymptomatic subcapsular hematomas resolve spontaneously and are conservatively managed, larger subcapsular hematomas may necessitate more focused intervention. Unbearable pain and renal compression and ischemia are indications for immediate intervention.

Both open surgical and percutaneous drainage methods for page kidney have been described, depending on the etiology of the compressing fluid or lesion and condition of the patient. Percutaneous drainage has been successfully used in the drainage of nonviscous or thick collections particularly pseudocysts [26]. Considering that the majority of percutaneous drains used for drainage of collections are relatively small, it is likely that hematomas are less likely to be drained effectively and quick enough to relieve the page kidney symptoms for which time is of the essence. Nevertheless, there are few published cases of successful percutaneous hematoma drainage in page kidney. Bansal et al. reported successful percutaneous drainage of a subcapsular hematoma secondary to ureteroscopy [8]. Similarly, Suckling et al. described clinically successful ultrasoundguided percutaneous drainage of a subcapsular hematoma with an 8F catheter in a 36-year-old female on warfarin therapy with a spontaneous case of page kidney [27]. The other disadvantage of percutaneous drainage, in theory, is the persistent presence of a compressive fibrotic pseudocapsule, which will continue to exert persistent compressive effect [28].

Therefore, prompt surgical evacuation should be considered the intervention of choice, especially in rapidly progressing hematomas, or when vital signs deteriorate [8,29]. Removal of the renal capsule and the constricting fibrous capsule has been shown to be curative [28]. Chung et al. had successful treatment of hematoma by evacuation in all cases of page kidney in a retrospective study of 519 patients after renal biopsy [15]. Our patient was expeditiously managed as such with good results.

Besides hematoma, the differential diagnosis for a subcapsular collection on ultrasound should also include abscess, urinoma, and lymphocele. An abscess most commonly presents as a thick-walled complex collection with heterogeneous echogenicity on ultrasound. There may be increased vascularity at the periphery of the collection due to inflammation. CT will often demonstrate a peripherally enhancing, medium density collection, possibly with septations or loculations [30]. On MRI, abscesses typically demonstrate T1 signal, intermediate to high T2 signal and possibly layering T2-hypointense debris [30,31]. Urinomas and lymphoceles will more closely mimic simple fluid and be anechoic on ultrasound, and may be more difficult to distinguish between [32]. Chylous lymphatic fluid may appear more echogenic than urine on ultrasound, and demonstrate layering fat [33]. Page kidney is an important post renal biopsy complication that should be recognized promptly to enable early intervention to preserve kidney function and secondary effects of malignant hypertension.

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