Acute Page kidney immediately following blunt trauma to a solitary pediatric kidney

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Abstract

Page kidney refers to the occurrence of hypertension secondary to renal compression and is usually associated with a subcapsular or perinephric hematoma. It generally occurs weeks to months after the initial injury. We report on a case of Page kidney occurring acutely after Grade IV blunt renal trauma in a pediatric patient with a solitary kidney following a tobogganing accident. The child was initially managed conservatively and discharged after six days bedrest. He re-presented post-injury Day 12 with recurrent hematuria, anemia, hypertension, and renal failure that required eventual, and successful, surgical exploration.

Introduction

Pediatric renal trauma management guidelines are based on the presence of two functioning kidneys and there is a paucity of literature describing management of trauma to a solitary kidney. Although expectant management is the cornerstone of managing most blunt renal trauma, a solitary kidney poses different challenges, as there is not a normal contralateral unit to provide physiologic compensation — a solitary Page kidney being one such example. While multiple guidelines state conservative management should be tried first in patients with renal trauma, in a pediatric patient with a solitary kidney, clinicians should have a low threshold for radiologic or surgical management if kidney perfusion and renal function start to decline.

Case report

A nine-year-old boy presented to the emergency department approximately eight hours after a tobogganing accident with blunt abdominal trauma and dark urine. A computed tomography (CT) scan of the abdomen and pelvis with and without contrast showed a Grade IV left renal laceration with associated left retroperitoneal hematoma, along with a congenital atrophic right kidney (Fig. 1). On admission, his blood pressure was 124/68 mmHg, pulse of 98, and a respiration rate of 24. His hemoglobin was 11g/dL and hematocrit was 35%.

Hospital admission 1

In the emergency department, the patient received one unit of packed red blood cells (PRBC). A physical exam showed only abdominal tenderness and guarding. A focused assessment with sonography for trauma (FAST) exam was negative. Repeat hemoglobin and hematocrit were 11g/dL and 33%, respectively. He was hospitalized for six days, during which his blood pressures ranged from 96–134/43–88 mmHg. His hematocrit decreased to 22.3%, but stabilized at 26% on discharge. His renal function and vital signs were stable throughout the hospitalization.

Hospital admission 2

The patient re-presented to the emergency department six days after discharge with with recurrent gross hematuria and left upper-quadrant abdominal pain. He did not report any additional trauma. His blood pressure was 145/80 mmHg with a creatinine of 0.9 mg/dL (increased from a baseline creatinine of 0.6 mg/dL), hematocrit of 23.5%, hemoglobin 8 g/dL, and a white blood cell count (WBC) of 18.9/uL. An abdominal ultrasound showed the laceration to the lower pole of the left kidney with decreased blood flow and an organized hematoma (Fig. 2). There was also a large blood clot seen in the patient's bladder.

On the next hospital day, his hematocrit dropped to 20.2%, WBC increased to 21.9/uL, creatinine elevated to 1.7mg/dL, and blood urea nitrogen (BUN) increased to 30 mg/dL. He was transfused one unit of PRBC. A Doppler ultrasound was repeated and showed an increasing large heterogeneous, nonvascular collection surrounding his left kidney involving the lower pole (Fig. 3). The left renal artery had a peak systolic velocity was 135 cm/s at the aorta and this increased to 523cm/s at the renal hilum (Figs. 4-5).

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Fig. 1. Outside computed tomography scan of abdomen and pelvis of a Grade IV renal laceration with a large retroperitoneal hematoma and an atrophic right kidney.

Fig. 2. Organized perinephric hematoma surrounding the left kidney.

At the hilum, there was also reversal of blood flow during diastole. Resistive indices (RI) at the interpolar and lower-pole arteries of the left kidney measured 1.0 compared to the upper pole RI of 0.68 (Fig. 6). In addition, the left renal vein showed loss of normal phasicity (Fig. 7).

As the patient was clinically deteriorating and there was concern for permanent renal functional loss, interventional radiology vs. surgical exploration was debated by the treatment team. Angiography with embolization of a bleeding segmental vessel +/- perinephric drainage of hematoma was considered a viable option, but concerns were that the patient would receive a contrast load, the vessel might not be acutely bleeding, and the perinephric hematoma might be too organized to effectively drain.

The patient, therefore, underwent an exploratory laparotomy. Via upper vertical midline incision, early renovascular control was achieved by a transmesenteric approach. Gerota's fascia was then opened and noted to be thick and contracted in the form of a reactive rind. Upon exposure of the left kidney, renal debridement and renorrhapy of the lower pole of the left kidney was performed, with five minutes of cross-clamp time. After organized clot evacuation, arterial bleeding was found from a lower-pole segmental artery supplying a devitalized segment, which was subsequently ligated. An intraoperative renal vascular Doppler was performed immediately post-debridement; the interpolar RI was 0.61 and the inferior portion of the interpolar RI was 0.47 (Fig. 8). The renal vein demonstrated better phasicity, with a peak velocity of 20 cm/s (Fig. 9). In addition, the renal artery velocity at the hilum declined to 56 cm/s (Fig.10).

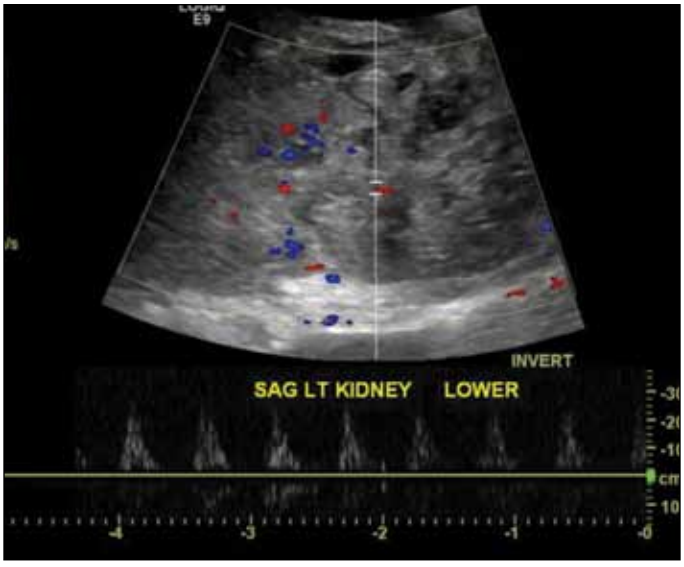

Fig. 3. Lack of diastolic flow to the lower pole of the left kidney.

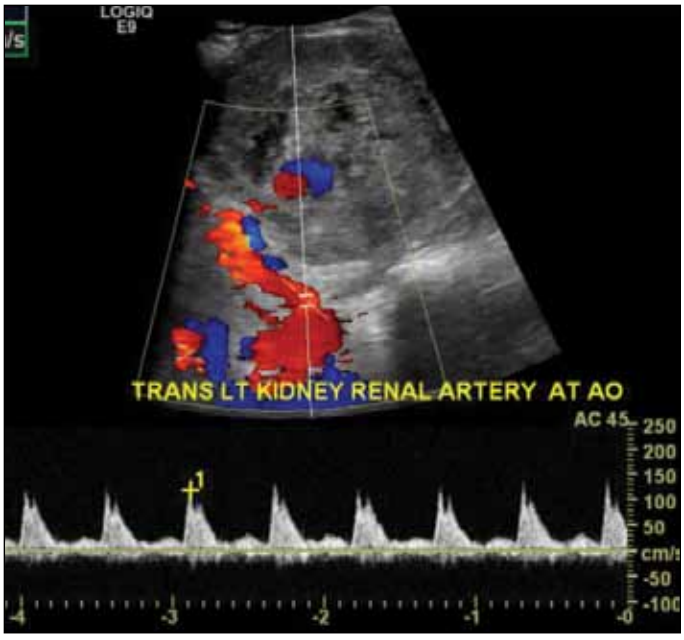

Fig. 4. Increased renal artery velocity at the aorta.

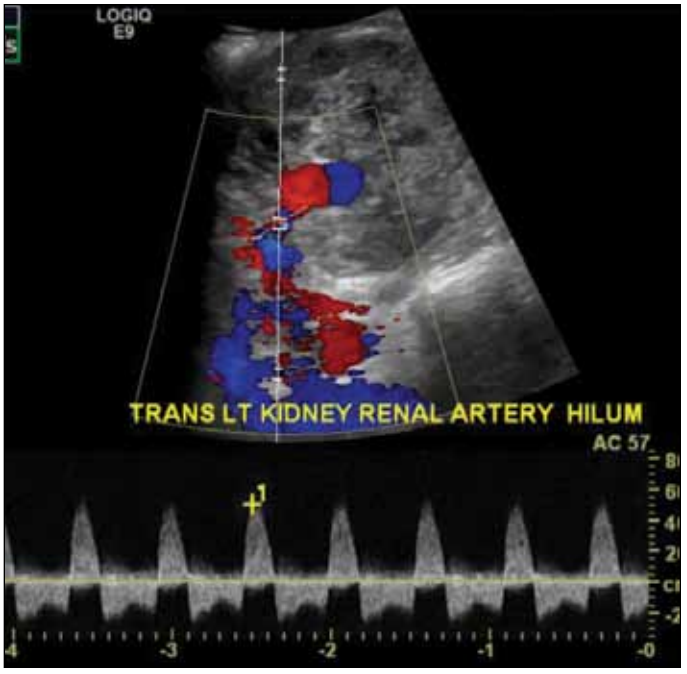

Fig. 5. Extremely increased renal artery velocity at the renal hilum with reversal of diastolic flow.

On postoperative Day 1, the patient's blood pressure was 118/69 mmHg and continued to improve to 106/78 at discharge. The patient's WBC trended down to 8.9/uL, his hematocrit stabilized at 29.2%, and his creatinine and BUN normalized to 0.6mg/dL and 14mg/dL, respectively at discharge. An ultrasound one month postoperative displayed resolution of the perinephric hematoma and fluid collection (Fig. 11).

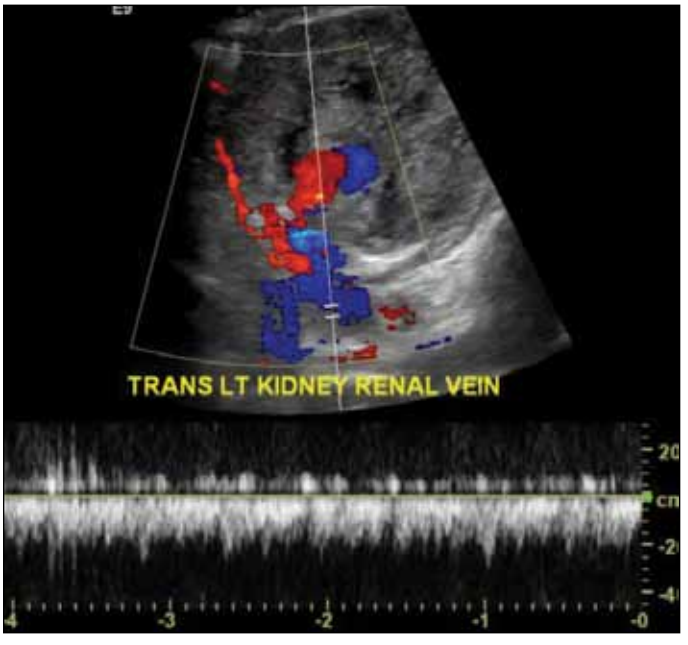

Fig. 7. Lack of pulsatile phasicity of the left renal vein.

Fig. 6. Lack of power Doppler located in the lower pole of the left kidney.

Discussion

The majority of Page kidneys develop in young men who experience blunt renal trauma. Page first described the pathophysiology in 1939.[1] Hypertension develops due to external

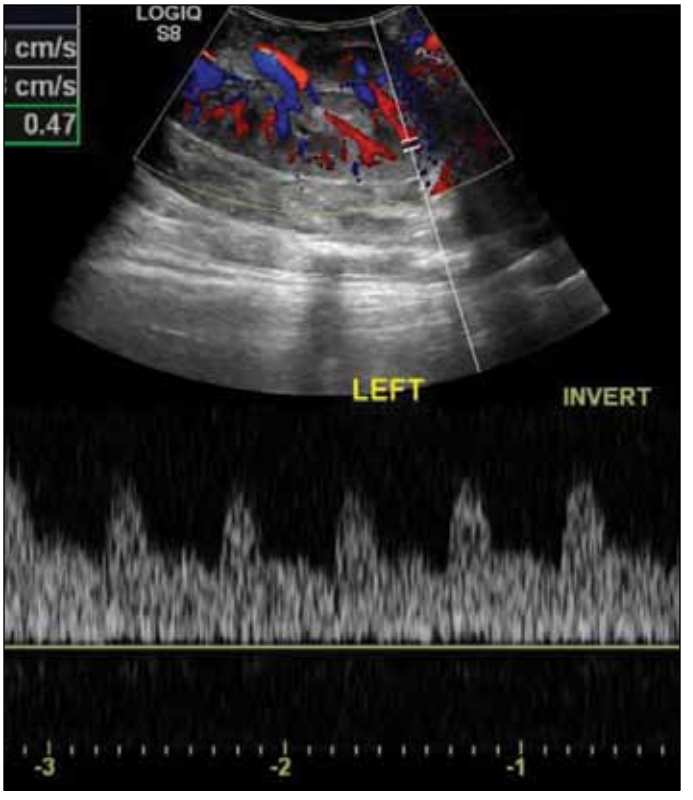

Fig. 8. Normalization of renal artery resistive indices post-surgical debridement with normalization of systolic and diastolic flow.

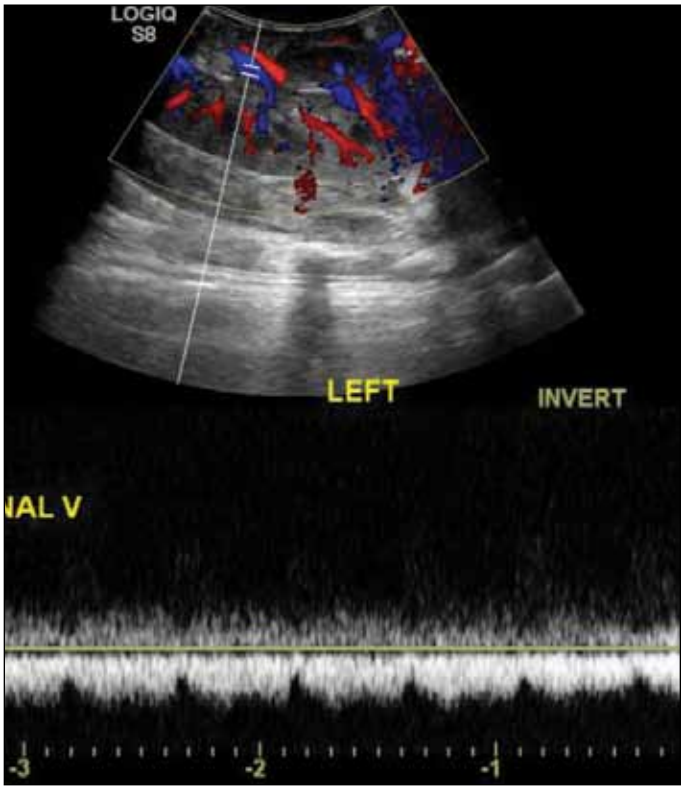

Fig. 9. Left renal vein post-debridement depicting normal venous pulsatile flow.

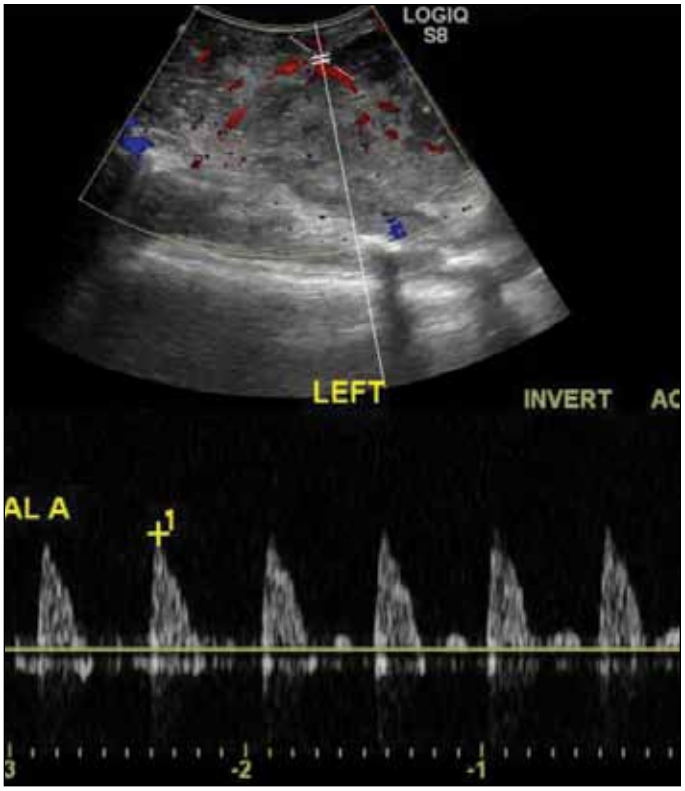

Fig. 10. Normalization of renal artery velocity post-surgical debridement with normal systolic and diastolic flow.

compression of renal parenchyma, leading to microvascular ischemia and activation of the renin-angiotensin-aldosterone system. Acute renal failure can develop if the contralateral kidney is diseased or nonfunctioning.

The American Urological Association (AUA) guidelines state that clinicians should use non-invasive management strategies in hemodynamically stable patients with renal injury (Grade B).[2] Similarly, the European Association of Urology (EAU) guidelines also state the majority of injured kidneys can be managed conservatively unless hemodynamically unstable or if the injury is a Grade V renal injury (Grade B and A, respectively).[3] Our patient was hemodynamically stable on presentation and was thus managed conservatively with blood transfusions and bed rest.

On his return presentation, he re-bled almost two weeks after the initial trauma. Reaction in Gerota's fascia created a thick and fibrous pseudocapsule around the kidney, leading to renal parenchymal compression from the hematoma. As a result, the RI increased significantly in the lower pole of the left kidney, resulting in a lack of perfusion. The retroperitoneal hematoma was compressing the renal artery, as suggested by reversal of diastolic blood flow and high renal artery velocities four times greater at the renal hilum than at the aorta. These ultrasound findings, along with persistent hypertension and acute renal failure, drove the decision towards surgical intervention. After surgical decompression, there was instantaneous normalization of RIs throughout the left kidney and in renal artery velocity.

In a similar case of recurrent, solitary Page kidney,[4] initial conservative radiologic management via percutaneous drainage of the subcapsular renal hematoma (along with sclerosing attempts) failed, leading to eventual definitive management requiring embolization. Success of percutaneous drainage of a hematoma may depend on its age and

Fig. 11. Ultrasound of the left kidney one month post-surgery.

liquefaction.[5] Laparoscopic evacuation can be also be performed depending on the clinical situation and surgeon's experience.[5]

Conclusion

Over time, urological trauma guidelines have evolved towards conservative management of renal trauma for parenchymal preservation. However, in a pediatric patient with a solitary kidney, precaution should be taken in monitoring adequate kidney perfusion and function. If conservative measures fail, as they did in this particular case, urologists should not hesitate in treating these patients either radiologically or surgically.

Competing interests: The authors declare no competing financial or personal interests.

This paper has been peer-reviewed.

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