Renal artery dissection as an overuse Injury

Sierra Grasso[1], Mia Laurel[2], Joseph Lewis[1], Mohammad Naiyer[1] ⓘD,
Richard Ricca[1] and George Keckeisen[1]

Abstract

The diagnosis of renal infarction is often convoluted due to its non-specific presentation. It can mimic disease processes as disparate as pyelonephritis, diverticulitis, or nephrolithiasis. This case is further complicated by the presence of a pelvic kidney with triplicate arterial input. It is difficult to estimate the incidence of pelvic kidneys as the numerous sources vary wildly in their estimations; however, the paucity information, in and of itself, speaks to the rarity of the condition. In this case, a 58-year-old male presents to the emergency department after experiencing sharp, sudden, and severe groin pain while swinging a golf club. The patient was noted to have an abnormally high systolic blood pressure in the 170s and hematuria, but all other initial labs and assessments were unremarkable. An initial computed tomography scan with intravenous contrast of the abdomen and pelvis showed partial necrosis of a pelvic kidney. Follow-up computed tomography angiography revealed that a dissection in one of the arteries supplying the kidney created an infarction and resultant necrosis. Vessel size, location and time between injury and diagnosis made endovascular intervention impractical. The patient was started on aspirin and Plavix, observed for 3 days and sent home.

Introduction

Renal infarction is an often overlooked diagnosis that is either missed or delayed with an incidence of approximately 0.004% in emergency department patients.[1] The main causes include arrhythmias and hypertension, hypercoagulable state, direct injury to the renal arteries, connective tissue disorders, or idiopathic.[2] Its presentation shares many commonalities with pyelonephritis, nephrolithiasis, or even diverticulitis.[13] There are numerous non-descript presenting signs and symptoms of a renal infarction such as abdominal, flank or groin pain, fever, nausea and vomiting, hypertension, hematuria and leukocytosis.[2] Two additional labs can be useful in screening for this diagnosis; lactate dehydrogenase (LDH) to illustrate tissue ischemia and c-reactive protein (CRP), which is an independent risk factor for the development of acute kidney injury (AKI). In addition, CRP is directly linked with infarction, leukocytosis and the presence of renal impairment.[4,5,2] However, this does not imply that elevations in these values lead to long-term chronic kidney disease. In fact, dissection of the renal artery does not appear to have any long-term effects on renal function.[6]

The second most common cause of renal infarction is renal artery dissection which accounts for 17.1%, following thromboembolic events.[6] Renal dissection most commonly occurs in men in their fourth decade of life. The most common risk factor is hypertension, followed by renal vascular injury and thromboembolic disease.[2] There is also some increased risk of dissection in connective tissue disorders, but this tends to play a smaller role.[7] Extreme exertion accompanied with sudden, explosive muscle contraction can lead to spontaneous dissection in adjacent vasculature due to shearing of the vessel wall.[8]

In their normal location kidneys lie within the lower aspect of the rib cage and are isolated from this type of insult.

[1]Department of Surgery, Stony Brook Southampton Hospital, Southampton, NY, USA
[2]Family Medicine & Neuromusculoskeletal Medicine, Stony Brook Southampton Hospital, Southampton, NY, USA

Corresponding Author:
Sierra Grasso, Department of Surgery, Stony Brook Southampton Hospital, 240 Meeting House Ln, Southampton, NY 11968, USA.
Email: sierra.grasso@gmail.com

In contrast, a pelvic kidney is surrounded by multiple large muscle groups and experiences recurrent compressive force from the bladder. In addition, in our patient, the dissected vessel is proportionally shorter than the other two arteries and may have experienced repeated increased tension leading to decreased vessel wall integrity.[8]

Renal ectopia results when the embryologic kidneys fail to ascend into the retroperitoneal renal fossa.[11] If the kidney fails to rise above the brim of the pelvis, it is considered a pelvic kidney.[12] The arterial supply to an ectopic kidney is highly variable because fetal vessels may fail to involute. It is not uncommon for ectopic kidneys to have double or triple arterial flows.[14] The arteries supplying pelvic kidneys may arise from the iliac arteries, aorta, hypogastric arteries or middle sacral arteries.[9] When the single arterial supply to a normal kidney becomes compromised, there is no compensatory anastomotic flow to protect against ischemia. Conversely, ectopic kidneys, that may have from one to three arteries, may retain arterial flow even if one artery is compromised.[10]

Case report

A 58-year-old male with a distant history of inguinal hernia repair presented to the emergency department with severe, sudden onset left groin pain after swinging a golf club. The pain was aggravated by lying or sitting, and not alleviated by over the counter strength acetaminophen. Vital signs on presentation were within normal limits except for a systolic blood pressure that remained in the 170s until midway through his hospital stay. On physical exam, the patient was positive for tenderness in the left lower quadrant, but negative for rebound pain. His bowel sounds were of normal quantity and quality, and clearly auscultated throughout the abdomen. Initial laboratory results, which are detailed in Table 1, were grossly normal. Ultrasound imaging was negative for testicular torsion or mass. A computed tomography (CT) scan of the abdomen and pelvis with intravenous (IV) and oral contrast revealed the presence of a pelvic kidney. Acute and chronic infarctions were noted in distant locations within that kidney.

The etiology of the infarction remained unknown. A full hypercoagulability panel, renal doppler ultrasound, and computed tomography angiography (CTA) were ordered along with initiation of a heparin drip for a presumed acute embolic event. The hypercoagulability panel (Table 2) and renal artery dopplers did not identify a cause for the infarction. The CTA showed three arterial supplies from the following locations: (1) the bifurcation of the aorta (implicated in the infarction); (2) the proximal portion of the right common iliac; and (3) the left internal iliac artery. A dissection was identified in the aortic branch supplying the anterior pole of the pelvic kidney and its associated necrosis (Figure 1–3).

Conversations with vascular surgery, interventional radiology and cardiology led to a decision to treat the infarction medically with dual antiplatelet therapy (Plavix 75 mg and

Table 1. Patient's laboratory values on hospital admission.

Laboratory Parameter	Value
Serum sodium	138 (136–145 mmol/L)
Serum potassium	4.2 (3.4–5.1 mmol/L)
Blood urea nitrogen	16 (8–23 mg/dL)
Serum creatinine	0.78 (0.70–1.20 mg/dL)
Serum lipase	18 (13–60 units/L)
Erythrocyte sedimentation rate	1 (0–15 mm/h)
C-reactive protein	0.11 (0.00–0.50 mg/dL)
White blood cell count	10.5 (4.2–11.0 ×10^9/L)
Red blood cell count	4.92 (4.06–5.63 ×10^{12}/L)
Hemoglobin	14.7 (12.5–16.3 g/dL)
Hematocrit	43.0 (36.7%–47.1%)
Platelets	258 (150–400 ×10^9/L)
Neutrophils	7.8 H (1.5–7.0 ×10^9/L)
Lymphocytes	1.3 (1.0–3.3 ×10^9/L)
Monocytes	0.7 (0.2–0.9 ×10^9/L)
Eosinophils	0.0 (0.0–0.6 ×10^9/L)
Basophils	0.1 (0.0–0.2 ×10^9/L)
Urine red blood cells	6-10 AB

Table 2. Patient's blood coagulation profile.

Laboratory parameter	Value
Prothrombin time	11.0 (10.0–12.9 s)
International normalized ratio	1.0 (0.8–1.2)
Fibrinogen	369 (275–515 mg/dL)
Thromboplastin time	39.7 H (26.9–38.8 s)
Protein S activity	96 (70%–180%)
Protein C activity	102 (70%–150%)
Antithrombin III activity	100 (80%–120%)
Lupus anticoagulant screen with dilute Russell's viper venom time	31 (<=45 s)
Factor V mutation	Not present (not applicable)

aspirin 325 mg) along with continuation of his heparin drip while hospitalized. An endovascular attempt to stent the dissected area was not plausible for several reasons. The size and location of the vessel made it difficult to access and may have caused more trauma to the area without overall benefit. In addition, the IV contrast needed to perform the procedure may have damaged the remaining healthy nephrotic tissue. After three hospital days, the patient was discharged in a hemodynamically stable condition, with instructions to continue the dual antiplatelet therapy medications for 6 months. Follow-up CT scan and labs showed a stable infarct and normal creatinine level indicating there was no lasting damage from the incident.

Discussion

Acute renal infarction is classically due to five underlying causes: arrhythmias and hypertension, hypercoagulable

Figure 1. Dissection in the arterial branch from the aortic bifurcation to the pelvic kidney.

Figure 2. Second cross-section of the dissection of the aortic branch to the pelvic kidney.
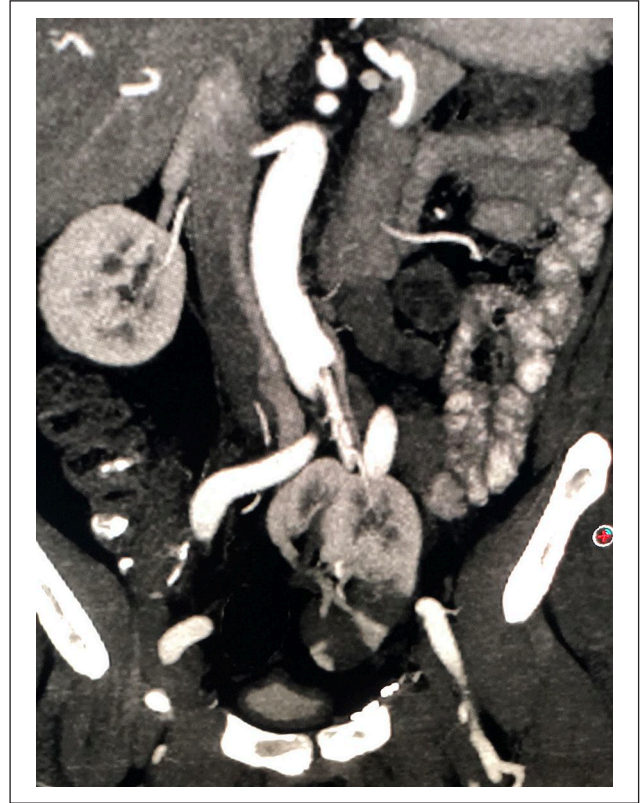

Figure 3. Alternative imaging window of the dissection of the aortic branch to the pelvic kidney.

state, direct injury to the renal arteries, connective tissue disorders, or idiopathic.[7,5] Our patient's infarction was the result of a dissection, believed to be the result of repeated trauma to the supplying artery. In the case of a complete renal artery dissection typical findings would include an elevated LDH and leukocytosis along with the other classic signs of hypertension, pain and hematuria.[2] In this case, despite the fact that the vessel arising from the aortic bifurcation was fully occluded, it behaved as a segmental branch because of the redundant arterial supply to the ectopic kidney. Only 20%–30% of the total renal volume of the left kidney was compromised so the resultant lab values appeared normal.[6]

One of the main predictors of renal infarction is prior renal infarction.[2] For this patient, the initial CT scan showed a small area of chronic infarction in the superior pole of the pelvic kidney. Closer investigation showed that this area was actually supplied by a limb of the dissected artery.[3] This common arterial origin hints that there is some form of underlying pathology unique to this particular artery. Review of the CT scan identified the implicated artery was also the shortest, and as a result, it may have experienced a greater amount of tension when the body moved through normal ranges of motion. Neither of the other two arteries supplying the damaged left kidney, nor the right renal artery supplying the uninjured kidney,

showed evidence of dissection or infarction. The patient had no evidence of thrombophilia, connective tissue disorder, or cardiac risk factors such as hypertension or arrhythmias. Therefore, it is reasonable to consider recurrent shear and compressive forces predisposed the affected artery to mechanical failure resulting in a dissection.

Ultimately, the ramification of the triplicate arterial flow confounded laboratory values that were normal. In a typical kidney with a full dissection and infarction of the supplying artery the CRP, blood urea nitrogen (BUN) and creatinine levels would be markedly elevated. In this case, the affected branch (aortic bifurcation origin) only represented 20%–30% of the blood supply to the pelvic kidney. The remaining viable renal tissue in the pelvis and normal anatomically positioned kidney was sufficient to mask evidence of renal compromise. Neither the position of the kidney in the pelvis, nor the delayed diagnosis changed any appreciable end result, either in the treatment, or final outcome of this patient. At the time of follow-up, the portion of the infarcted kidney was stable, and his renal function was within normal limits. This is consistent with the typical outcome of segmental renal artery dissection in the normal anatomic location.[6]

Conclusion

In every way, this patient mirrored a similar presentation to that of a segmental renal artery dissection in an anatomically normal kidney, complete with lab values, presentation and outcome. This was possible due to the triplicate blood supply where only one branch was affected. The significant difference here is the reason for dissection. The path of the vessel through the body cavity on CTA illustrates a vessel under tension due to its shorter length that ultimately failed. The length alone may not have been an issue, but the position of the pelvic kidney placed the vessel adjacent to other vasculature, pelvic musculature, and organs. The additive forces likely led to the dissection.

Declaration of conflicting interests

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

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

ORCID iD

Mohammad Naiyer [iD] https://orcid.org/0000-0002-2969-606X

References

1. Huang CC, Lo HC, Huang HH, et al. ED presentations of acute renal infarction. *Am J Emerg Med* 2007; 25(2): 164–169.
2. Saeed K. Renal infarction. *Int J Nephrol Renovasc Dis* 2012; 5: 119123.
3. Zhou H, Yan Y, Li C, et al. Acute thrombo-embolic renal infarction. *Urol Case Rep* 2016; 7: 31–32
4. Antopolsky M, Simanovsky N, Stalnikowicz R, et al. Renal infarction in the ED: 10-year experience and review of the literature. *Am J Emerg Med* 2012; 30(7): 1055–1060
5. Bourgault M, Grimbert P, Verret C, et al. Acute renal infarction: a case series. *Clin J Am Soc Nephrol* 2013; 8(3): 392398.
6. Yoon K, Song SY, Lee CH, et al. Spontaneous renal artery dissection as a cause of acute renal infarction: clinical and MDCT findings. *J Korean Med Sci* 2017; 32(4): 605–612
7. Emanuela C, Francesco C, Massimiliano PA, et al. Spontaneous renal artery dissection in ehler-danlos syndrome. *Kidney Int Rep* 2019; 4(11): 1649–1652.
8. Renaud S, Leray-Moraguès H, Chenine L, et al. Spontaneous renal artery dissection with renal infarction. *Clin Kidney J* 2012; 5(3): 261–264.
9. Eid S, Iwanaga J, Loukas M, et al. Pelvic kidney: a review of the literature. *Cureus* 2018; 10(6): e2775.
10. Charles Brunicardi F, Andersen D, Billiar T, et al. *Schwartz's Principles of Surgery*. 10th ed. NewYork: McGraw-Hill Education / Medical, 2014, pp. 1651.
11. Rosenblum ND. Renal ectopic and fusion anomalies: renal ectopy. In: KT Mattoo, LS Baskin and MS Kim (eds) *Uptodate, Topic 6107* (Version 15.0), Waltham, MA: Uptodate 2020.
12. Bhoil R, Sood D, Singh YP, et al. An ectopic pelvic kidney. *Pol J Radiol* 2015; 80: 425–427.
13. Piccoli GB, Priola AM, Vigotti FN, et al. Renal infarction versus pyelonephritis in a woman presenting with fever and flank pain. *Am J Kidney Dis* 2014; 64(2): 311–314.
14. Gencheva R, Gibson B, Garugu S, et al. A unilateral pelvic kidney with variant vasculature: clinical significance. *J Surg Case Rep* 2019; 2019(11): rjz333.