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Renovascular Hypertension Associated With Pseudoaneurysm Following Blunt Trauma

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Abstract

We present the case of a 21-year-old man who developed a renal artery pseudoaneurysm following a 7-foot fall onto his back. He initially presented with gross hematuria, left flank pain, and back pain. He was observed in the hospital for 3 days and discharged. One week later, he was readmitted with headache, nausea, vomiting, seizure activity, and hypertension. Contrast-enhanced computed tomography of the abdomen showed a left renal artery pseudoaneurysm with associated arterial narrowing and delayed ipsilateral renal enhancement. He underwent percutaneous stent-graft placement with resolution of the pseudoaneurysm. He was free of complications and normotensive off antihypertensive medications after 36 months of follow-up. Renal artery pseudoaneurysms are rare and under-recognized complications of blunt abdominal or back trauma that can cause hypertension. Imaging modalities in renovascular hypertension have focused on detecting renal artery stenosis from atherosclerotic disease or fibromuscular dysplasia, with little attention given to renal artery pseudoaneurysms. In addition, first-line treatment for renal artery pseudoaneurysms historically has consisted of angioembolization, yet percutaneous stent-graft placement has emerged as an attractive alternative to preserve vessel patency. We discuss the role of imaging in renovascular hypertension with a focus on renal artery pseudoaneurysms and their prevalence, diagnosis, and treatment.

Keywords

Pseudoaneurysm; renovascular hypertension; imaging; blunt trauma; renal artery

INTRODUCTION

A true aneurysm consists of a dilation involving all 3 arterial wall layers (intima, media, and adventitia),¹ whereas a pseudoaneurysm begins as a contained hematoma formed after damage to at least one of the arterial wall layers.² Reactive fibrosis occurs in the surrounding connective tissue encapsulating the hematoma, which forms a structure resembling an aneurysmal sac. A pseudoaneurysm is a serious condition because if the blood clot at the site of the arterial injury dissolves and re-establishes communication with the arterial lumen, this can result in pseudoaneurysmal expansion or rupture.²

Although more rare than fibromuscular dysplasia and atherosclerotic-related renal artery stenosis,³ a renal artery pseudoaneurysm can also lead to renovascular hypertension.⁴ There have been numerous studies assessing the imaging modalities used to evaluate renovascular hypertension from atherosclerotic disease or fibromuscular dysplasia.^{5–11} This report

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discusses the advantages and limitations of renal artery imaging by describing a case of acute hypertension following a renal artery pseudoaneurysm caused by blunt trauma.

CASE REPORT

Clinical History and Initial Laboratory Data

A previously healthy 21-year-old man presented to a local hospital with left flank pain, back pain, and gross hematuria after falling 7 feet onto his back. He was admitted with blood pressure (BP) of 145/96 mm Hg and observed in the hospital for 3 days. Computed tomography (CT) revealed a left lower pole renal infarction (Fig 1C). One week after discharge, he awoke with a throbbing retro-orbital headache followed by nausea, vomiting, visual disturbances, and brief seizure activity.

At readmission, the patient had BP of 175/108 mm Hg with otherwise normal vital signs. Findings from physical examination, including a complete neurologic evaluation, were unremarkable. Laboratory studies showed serum urea nitrogen level of 9 mg/dL; creatinine level of 1.2 mg/dL, corresponding to estimated glomerular filtration rate of 86 mL/min/1.73 m² as calculated by the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equation¹²; and hemoglobin level of 15.4 g/dL. Urinalysis showed 3-10 red blood cells per high-power field and protein (1+). He was given fosphenytoin and labetalol and had no recurrence of seizure activity.

Imaging Studies

CT of the abdomen and pelvis with iodinated intravenous contrast showed a 2.1×1.5-cm left renal hilar mass obstructing the left renal artery with associated segmental infarctions of the left kidney (Fig 1B and D). The distal aspect of the pseudoaneurysm was mildly narrowed by the associated hematoma. Comparison with computed tomographic imaging from 10 days prior showed that the hilar mass had progressed (Fig 1A and B). Given the rapid evolution of this “mass” in the setting of recent trauma, a vascular injury was suspected. A renal arteriogram showed 2 renal arteries on each side, and a pseudoaneurysm was identified in the midportion of the superior left renal artery with associated filling defect at its distal margin (Fig 2A). A stent-graft was placed with no residual pseudoaneurysm and good blood flow to the superior pole of the kidney (Fig 2B).

Diagnosis

Renal pseudoaneurysm–associated renovascular hypertension.

Clinical Follow-up

The morning after stent-graft placement, the patient’s BP was 157/99 mm Hg. At 1-month follow-up, he was asymptomatic and office BP was 111/67 mm Hg with heart rate of 69 beats/min while on treatment with labetalol, 400 mg, twice daily. Laboratory studies showed serum urea nitrogen level of 7 mg/dL, creatinine level of 1.1 mg/dL (estimated glomerular filtration rate, 96 mL/min/1.73 m²), hemoglobin level of 14.6 g/dL, and urine protein-creatinine ratio of 0.07 mg/mg. A subsequent Doppler ultrasound study showed patent left renal arteries with normal velocities. BP medication was tapered off during the next month and office BPs were 115/84, 123/76, and 119/78 mm Hg at 6, 18, and 36 months, respectively. Three years later, CT angiography of the abdomen was performed, showing a patent stent-graft without stenosis or aneurysm.

DISCUSSION

Renal artery pseudoaneurysms occur as a result of trauma or iatrogenic injury. Iatrogenic causes are uncommon, with documented occurrence rates of 1%-3.7% following renal artery angioplasty with or without stent placement.^{13,14} Penetrating abdominal trauma has the highest reported incidence of pseudoaneurysm formation at 9.7% after stab wounds involving the kidney.¹⁵ Formation of a renal artery pseudoaneurysm after blunt trauma to the back, flank, or abdomen is less common: in 2001-2007, only 18 cases were reported in the urologic literature.¹⁶

Renal pseudoaneurysms typically present as flank pain, hematuria, and/or unexplained anemia after back or abdominal trauma.² In cases of blunt trauma, presentation can be delayed days to years after the injury, with the longest reported delay being 15 years.¹⁷ This delay likely is explained by the new rapid expansion of a smaller, unrecognized, and previously contained pseudoaneurysm. Rupture of a renal artery pseudoaneurysm can result in hypotension from acute hemorrhage; however, stable or enlarging pseudoaneurysms may impair renal blood flow and produce renin-mediated hypertension.

In our case, the patient's initial hematuria and flank pain likely resulted from renal arterial wall injury leading to renal infarction. The renal artery injury initially was below the resolution of computed tomographic imaging (Fig 1A). One week later, abdominal CT revealed a renal artery pseudoaneurysm (Fig 1B), suggesting that a smaller pseudoaneurysm had expanded. Disrupted renal artery blood flow from the expanding pseudoaneurysm with partial thrombosis could explain the hypertension and associated neurologic manifestations.

Decreased kidney perfusion from renal artery stenosis is a known cause of hypertension, termed renovascular hypertension; however, renal artery stenosis is not required to cause renin-mediated hypertension. Disrupted blood flow from a pseudoaneurysm would have the same downstream turbulence and low resistance that typically would trigger hypertension from a poststenotic artery in the setting of renal artery stenosis. This effect is highlighted in our case, in which no obvious poststenotic dilatation is seen in the renal arteriogram (Fig 2A). However, evidence of reduced renal blood flow is confirmed by the delayed enhancement of the left kidney on the CT angiogram (Fig 1B and D). Delayed renal enhancement is seen only with hemodynamically significant reductions in renal blood flow.

Because a thrombus does not enhance on catheter arteriography or CT angiography, a pseudoaneurysm composed primarily of clot can be underestimated in size. The small appearance of the pseudoaneurysm in our case is due partially to thrombus formation (seen as lack of enhancement in Fig 1B and the hypodensity in the distal pseudoaneurysm on Fig 2A). However, findings on the CT angiogram confirm that the pseudoaneurysm is small. Therefore, the size of the pseudoaneurysm on renal arteriography or CT angiography cannot predict renin-mediated hypertension from renal arterial blood flow interruption.

Contrast-enhanced CT of the abdomen often is the initial imaging study in the diagnosis of a renal artery pseudoaneurysm. On unenhanced images, pseudoaneurysms may be difficult to differentiate from a cyst or hematoma and therefore are best identified using contrast-enhanced studies. High-resolution arterialphase images often are used to optimize visualization of the arterial structures, including a pseudoaneurysm. On contrasted images, a pseudoaneurysm is identified as a focal rounded area of contrast enhancement equal in attenuation to adjacent arterial vessels.¹⁸

Duplex ultrasonography, captopril isotope renography, CT angiography, magnetic resonance angiography, and catheter-directed renal arteriography have all been used to detect abnormalities in kidney perfusion. Renal arteriography with digital subtraction and

transstenotic pressure gradient measurement for functional assessment of hemodynamic significance is the gold standard for diagnosing renal artery stenosis.⁵ A large number of studies have compared the accuracy of imaging modalities used to detect renovascular hypertension from renal artery stenosis.⁵⁻¹¹ Results from prospective studies since 2000 are summarized in Table 1.

Duplex ultrasonography provides a noninvasive hemodynamic means to evaluate arterial lesions and detect kidney size abnormalities.¹⁹ Peak systolic velocity >200 cm/s and a renal to aortic peak flow velocity ratio >3.5 are the most dependable ultrasound measurements to identify severe renal artery stenosis.²⁰ Color and spectral Doppler can detect pseudoaneurysm formation as a round structure with circular flow pattern and “to-and-fro” biphasic flow at the pseudoaneurysm neck, respectively. The neck often is the only patent portion of the pseudoaneurysm due to thrombosis. Thrombus in the pseudoaneurysm usually appears hypoechoic without flow; it typically is mural but may centrally fill a portion of the pseudoaneurysm lumen.¹⁸

Magnetic resonance angiography is useful as an adjunct to ultrasonography or an alternative in patients with a contraindication to CT contrast, such as iodine allergy or reduced kidney function. A pseudoaneurysm seen with magnetic resonance angiography will appear as a round focal enhancing structure with intensity similar to that of adjacent arteries. A mural thrombus may have high T1 signal intensity on unenhanced series.¹⁸

Choosing the initial imaging modality to evaluate renovascular hypertension depends on the clinically suspected cause and patient risk factors. In older patients in whom atherosclerotic-related renal artery stenosis is more common, duplex ultrasonography is a reasonable first choice. If there is a high clinical suspicion of renal artery pseudoaneurysm or fibromuscular dysplasia, renal arteriography should be an early consideration because endovascular intervention (stent-graft and balloon angioplasty, respectively) is effective treatment for renovascular hypertension. In our case, contrast-enhanced CT of the abdomen was followed directly by renal arteriography with stent-graft placement.

A stent-graft or covered stent is wrapped in a fabric-like material that is relatively nonporous.²¹ The stiff tube lies in communication with the arterial wall and when spanning an aneurysmal dilation, relieves the pressure on the weakened arterial wall. Stentgrafts are used most commonly to treat abdominal aortic aneurysms. Complications from endovascular renal artery stenting include embolization, acute stent thrombosis, stent fracture, renal artery spasm, rupture, perforation or dissection, acute kidney injury, and access-site morbidity.^{22,23} There are no published data for the long-term patency of stent-grafts used to treat renal vascular pseudoaneurysms.²⁴ However, renal artery stents have been used in conjunction with angioplasty to treat atherosclerotic renal artery stenosis and have 2-year in-stent restenosis rates of 29.7%.²⁵ Rarely, renal artery stent-related infections can occur (5 case reports), often identified by mycotic pseudoaneurysm formation on duplex ultrasonography.²⁶

In conclusion, a renal artery pseudoaneurysm is a potentially life-threatening condition necessitating early diagnosis and intervention. Its clinical presentation typically is delayed for days to years following renal artery injury. A high level of clinical suspicion is required to make the diagnosis. Patients with new-onset hematuria, flank pain, or abrupt changes in BP should be questioned about back/abdominal trauma, including blunt trauma or renal artery interventions. A positive history would prompt abdominal contrast-enhanced CT, with renal arteriography reserved for patients with kidney abnormalities on CT. When the anatomy is suitable, blood vessel-preserving treatment with a stent-graft is preferred to

angioembolization and can be curative for the associated renovascular hypertension. Patency of the renal artery stent-graft can be monitored effectively by serial duplex ultrasonography.

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REFERENCES

1. Sueyoshi E, Sakamoto I, Nakashima K, Minami K, Hayashi K. Visceral and peripheral arterial pseudoaneurysms. *AJR Am J Roentgenol.* 2005; 185(3):741–749. [PubMed: 16120928]
2. Ngo TC, Lee JJ, Gonzalgo ML. Renal pseudoaneurysm: an overview. *Nat Rev Urol.* 2010; 7(11): 619–625. [PubMed: 20938436]
3. Safian RD, Testor SC. Renal-artery stenosis. *N Engl J Med.* 2001; 344(6):431–442. [PubMed: 11172181]
4. Mehta AN, Fenves A. Current opinions in renovascular hypertension. *Proc (Bayl Univ Med Cent).* 2010; 23(3):246–249. [PubMed: 20671820]
5. Eklof H, Ahlstrom H, Magnusson A, et al. A prospective comparison of duplex ultrasonography, captopril renography, MRA, and CTA in assessing renal artery stenosis. *Acta Radiol.* 2006; 47(8): 764–774. [PubMed: 17050355]
6. Rountas C, Vlychou M, Vassiou K, et al. Imaging modalities for renal artery stenosis in suspected renovascular hypertension: prospective intraindividual comparison of color Doppler US, CT angiography, GD-enhanced MR angiography, and digital subtraction angiography. *Ren Fail.* 2007; 29(3):295–302. [PubMed: 17497443]
7. Vasbinder GB, Nelemans PJ, Kessels AG, et al. Renal Artery Diagnostic Imaging Study in Hypertension (RADISH) Study Group. Accuracy of computed tomographic angiography and magnetic resonance angiography for diagnosing renal artery stenosis. *Ann Intern Med.* 2004; 141(9):674–682. [PubMed: 15520423]
8. Solar M, Zizka J, Krajina A, et al. Comparison of duplex ultrasonography and magnetic resonance imaging in the detection of significant renal artery stenosis. *Acta Med (Hradec Kralove).* 2011; 54(1):9–12.
9. Fommei E, Ghione S, Hilson AJ, et al. European Multicentre Study Group. Captopril radionuclide test in renovascular hypertension: a European multicentre study. *Eur J Nucl Med.* 1993; 20(7):617–623. [PubMed: 8370384]
10. Johansson M, Jensen G, Aurell M, et al. Evaluation of duplex ultrasound and captopril renography for detection of renovascular hypertension. *Kidney Int.* 2000; 58(2):774–782. [PubMed: 10916102]
11. Vasbinder GB, Nelemans PJ, Kessels AG, Kroon AA, de Leeuw PW, van Engelshoven JM. Diagnostic tests for renal artery stenosis in patients suspected of having renovascular hypertension: a meta-analysis. *Ann Intern Med.* 2001; 135(6):401–411. [PubMed: 11560453]
12. Levey AS, Stevens LA, Schmid CH, Zet al, CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration). A new equation to estimate glomerular filtration rate. *Ann Intern Med.* 2009; 150(9):604–612. [PubMed: 19414839]
13. Tan J, Fillobbos R, Raghunathan G, et al. Efficacy of renal artery angioplasty and stenting in a solitary functioning kidney. *Nephrol Dial Transplant.* 2007; 22(7):1916–1919. [PubMed: 17400563]
14. ASTRAL Investigators. Wheatley K, Ives N, Gray R, et al. Revascularization versus medical therapy for renal-artery stenosis. *N Engl J Med.* 2009; 361(20):1953–1962. [PubMed: 19907042]
15. Heyns CF, Van Vollenhoven P. Increasing role of angiography and segmental artery embolization in the management of renal stab wounds. *J Urol.* 1992; 147(5):1231–1234. [PubMed: 1569655]
16. Lindeklev H, Hargo S, Nordhus K, Eggen T, Due J. Renal artery pseudoaneurysm secondary to blunt trauma nine years earlier: case report and review of the literature. *Scand J Urol Nephrol.* 2008; 42(5):488–491. [PubMed: 18609285]

17. Jebara VA, El Rassi I, Achouh PE, Chelala D, Tabet G, Karam B. Renal artery pseudoaneurysm after blunt abdominal trauma. *J Vasc Surg.* 1998; 27(2):362–265. [PubMed: 9510292]
18. Kapoor BS, Haddad HL, Saddekni S, Lockhart ME. Diagnosis and management of pseudoaneurysms: an update. *Curr Probl Diagn Radiol.* 2009; 38(4):170–188. [PubMed: 19464587]
19. Baumgartner I, Lerman LO. Renovascular hypertension: screening and modern management. *Eur Heart J.* 2011; 32(13):1590–1598. [PubMed: 21273200]
20. Zeller T, Bonvini RF, Sixt S. Color-coded duplex ultrasound for diagnosis of renal artery stenosis and as follow-up examination after revascularization. *Catheter Cardiovasc Interv.* 2008; 71(7): 995–999. [PubMed: 18383154]
21. Yeo KK, Rogers JH, Laird JR. Use of stent grafts and coils in vessel rupture and perforation. *J Intervent Cardiol.* 2008; 21(1):86–99. [PubMed: 18254790]
22. Mwipatayi BP, Beningfield SJ, White LE, et al. A review of the current treatment of renal artery stenosis. *Eur J Vasc Endovasc Surg.* 2005; 29(5):479–488. [PubMed: 15966086]
23. Schuurman JP, de Vries JP, Vos JA, Wille J. Renal artery pseudoaneurysm caused by a complete stent fracture: a case report. *J Vasc Surg.* 2009; 49(1):214–216. [PubMed: 19174257]
24. Horwitz MD, Hanbury DC, King CM. Renal artery pseudoaneurysm following partial nephrectomy treated with stent-graft. *Br J Radiol.* 2005; 78(926):161–163. [PubMed: 15681331]
25. Vignali C, Bargellini I, Lazzereschi M, et al. Predictive factors of in-stent restenosis in renal artery stenting: a retrospective analysis. *Cardiovasc Intervent Radiol.* 2005; 28(3):296–302. [PubMed: 15770391]
26. Stout CL, Glickman MH. Renal artery stent infection and pseudoaneurysm management. *Ann Vasc Surg.* 2010; 24(1):114.e13–e17. [PubMed: 19748220]

Figure 1.

Computed tomography angiography of the patient's abdomen at the time of injury and 8 days later. (A) A mildly dilated left superior renal artery without a distinct pseudoaneurysm (arrow) at the time of his fall. (B) Expansion of the renal artery 8 days later (arrow). (C) Left renal infarctions in the inferior pole (arrow) at the time of his fall. (D) Coronal image that has been magnified to highlight the delayed left renal enhancement and left renal infarction with a capsular rim sign (arrow).

Figure 2. Catheter angiography of the left superior renal artery. (A) Circumferential pseudoaneurysm with clot at the distal aspect (arrow). (B) Resolution of the pseudoaneurysm with stent-graft placement (arrow).

Table 1**Prospective Trials Comparing Imaging Modalities in Detecting Renal Artery Stenosis**

Modality and Trial	Sensitivity Specificity			
	N	(%)	(%)	Standard
Duplex ultrasonography				
Eklof et al ⁵ (2006)	58	80	54	DSA with PGM
Rountas et al ⁶ (2007)	58	75	90	DSA
Solar et al ⁸ (2011)	94	85	84	DSA
Johansson et al ¹⁰ (2000)	98	84	94	DSA
Captopril renography				
Eklof et al ⁵ (2006)	58	59	50	DSA with PGM
Johansson et al ¹⁰ (2000)	98	68	92	DSA
CTA				
Eklof et al ⁵ (2006)	58	100	56	DSA with PGM
Rountas et al ⁶ (2007)	58	94	93	DSA
Vasbinderet al ⁷ (2004)	356	64	92	DSA
MRA				
Eklof et al ⁵ (2006)	58	98	70	DSA with PGM
Rountas et al ⁶ (2007)	58	90	94	DSA
Vasbinder et al ⁷ (2004)	356	62	84	DSA
Solar et al ⁸ (2011)	94	93	93	DSA
DSA				
Eklof et al ⁵ (2006)	58	95	91	PGM 15 mm Hg

Abbreviations: CTA, computed tomography angiography; DSA, digital subtraction angiography; MRA, magnetic resonance angiography; PGM, trans-stenotic pressure gradient measurement.