



Spontaneous Renal Artery Dissection: A Case Report and Brief Review of the Literature

Tamara Wanigasekera¹, Rosemary Lucey¹, Ayanfeoluwa Obilana¹, Kevin O'Regan², Frank L. Ward^{1*}

¹Department of Nephrology, Cork University Hospital, Cork, Ireland.

²Department of Radiology, Cork University Hospital, Cork, Ireland.

***Corresponding Author:** Dr. Frank Ward, Department of Nephrology, Cork University Hospital, Wilton, Cork City, Ireland. Email: francis.ward@hse.ie

Abstract: Spontaneous renal artery dissection (SRAD) is a rare cause of renovascular hypertension. It represents a diagnostic challenge for physicians, requiring a high index of suspicion and lends itself towards a delayed diagnosis in many cases. The clinical presentation is non-specific, varying from incidental hypertension to flank pain and fever, with or without acute kidney injury. SRAD can lead to renal infarction in the distribution of the affected artery, resulting in chronic kidney disease and chronic hypertension. There remains no consensus on the optimal management. Multiple strategies have been described, including anti-hypertensive therapy and anticoagulation, with urgent endovascular or surgical revascularization warranted in the acute setting for deteriorating renal function or treatment of refractory renovascular hypertension. We present the case of a 37-year-old man who presented with acute left flank pain and hypertension due to unrecognized SRAD. Hypertension was controlled adequately with combination drug therapy. Magnetic resonance angiography was later electively performed as part of a secondary hypertension work-up, and revealed newly established renal infarction of the left kidney lower pole and inter-polar areas. A prompt diagnosis at the time of initial presentation may have allowed an opportunity for renal revascularization and possibly prevention of renal infarction.

Keywords: Renal artery dissection, renovascular hypertension, chronic kidney disease

1. INTRODUCTION

Spontaneous renal artery dissection (SRAD) occurs outside the setting of direct trauma or as an adverse event during endovascular intervention (e.g. renal artery angioplasty). The disease is rare, with approximately 200 cases reported in the literature, although this likely under-represents the true incidence. There is a strong male preponderance and presentation is typically in the third or fourth decade.¹ Underlying abnormalities within the vessel are a risk factor, associated with connective tissue disorders such as Ehlers Danlos and Marfan syndrome, atherosclerosis and fibromuscular dysplasia (FMD).²⁻⁴ SRAD is also reported in those with normal native vessels, with strenuous physical activity such as weight-lifting being reported as a precipitant in some instances.⁵ This suggests a possible pathogenesis relating to increased shear forces across the renal artery, possibly explaining why the shorter left renal artery is more commonly affected than the right.

SRAD presents a diagnostic challenge as the clinical presentation may be non-specific; often mimicking pyelonephritis or renal colic and significant findings may not be evident on routine ultrasound or computerized tomography (CT) imaging at the time of an acute presentation. Without a strong clinical suspicion to pursue further diagnostics, the diagnosis may be delayed or missed, leading to irreversible renal infarction and chronic hypertension in many cases.

2. CASE PRESENTATION

2.1. Clinical History and Initial Laboratory Data

A 37-year-old man presented to the Emergency Department with sudden onset, non-traumatic, left flank pain which occurred while sitting in a business meeting. He had severe hypertension, with an initial blood pressure of 195/112mmHg. The patient had undertaken a routine 24-hour ambulatory blood pressure monitor with his

family doctor three months earlier (due to a family history of diabetes mellitus), which demonstrated normotension at that time. He was taking no medications. Physical examination revealed a BMI of 36kg/m², but was otherwise unremarkable. Admission laboratory values included a normal serum creatinine of 1.06mg/dL and potassium of 4.4 mEq/L. Initial urinalysis was positive for albumin, with a spot urine protein/creatinine ratio of 1460mg/g. The plasma renin level was significantly elevated (732pg/ml), with a normal serum aldosterone level. A non-contrast enhanced CT scan of the abdomen revealed a normal renal tract and no obvious source of the flank pain. The flank pain subsequently resolved within 48-hours. Combination anti-hypertensive medication with olmesartan, amlodipine and hydrochlorothiazide achieved satisfactory blood pressure control. A work-up for secondary hypertension was initiated and the patient was referred for outpatient nephrology review.

2.2. Additional Investigations

Two months following the acute presentation, the patient underwent a magnetic resonance angiogram (MRA) of the kidneys (Fig. 1). The MRA revealed a left kidney supplied by two renal arteries, with a patent small upper pole branch and a lower pole branch dissected and occluded after 1.5cm (marked with arrow). The inter-polar and lower pole kidney parenchyma supplied by this occluded artery demonstrated cortical thinning and atrophy, consistent with renal infarction, which was new compared to CT imaging two months previously (Fig 2). There was a single normal appearing right renal artery, with no findings to suggest underlying FMD. A trans-thoracic echocardiogram was within normal limits, with no evidence of left ventricular hypertrophy.

2.3. Diagnosis

The clinical presentation and subsequent findings are consistent with a spontaneous left renal artery dissection.

2.4. Clinical Follow up

The patient's blood pressure remains currently well-controlled through diet and life-style modification and combination anti-hypertensive therapy. The renal function has remained stable, with a normal serum creatinine level and a normal urine protein/creatinine ratio. He also receives low-dose anti-platelet therapy and a statin.

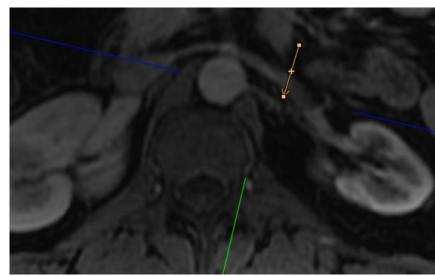


Figure1. Axial contrast-enhanced T1-weighted MR angiogram of the upper abdomen

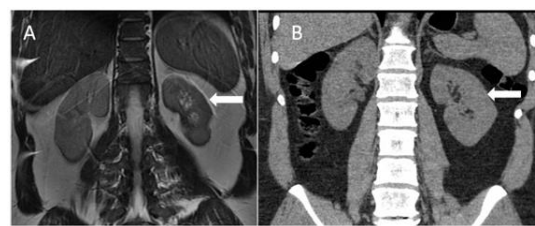


Figure2. (A) Coronal T2-weighted MRI of the abdomen, which demonstrates atrophy of the left kidney (arrow). Note the discrepancy in size compared to the normal right kidney. (B) Coronal non-contrast CT of the abdomen performed 2 months earlier, which demonstrates a normal left kidney (arrow).

3. DISCUSSION

Spontaneous (non-traumatic) renal artery dissection (SRAD) is a rarely reported phenomenon. Given the perceived low incidence and a clinical presentation that may be silent or resolve spontaneously (as in this case), the diagnosis represents a challenge to clinicians that requires a high index of suspicion and lends itself towards a delayed diagnosis. SRAD does not have a clear precipitant in many cases, but has been associated with strenuous physical exertion, atherosclerotic disease, malignant hypertension or an underlying abnormality of the blood vessel, such as FMD.

When clinical suspicion is aroused, CT or MR angiography is most commonly employed as the initial diagnostic test. Ultrasonography with Doppler is insensitive for detection of SRAD, due to its operator dependence and the fact that the dissection may be limited to the intra-renal arteries in some cases. Formal direct angiography is the gold standard for diagnosis and also allows for subsequent endovascular intervention.

Management of SRAD is dependent on the clinical presentation and timing of diagnosis. Successful outcomes are reported with conservative medical management, including anti-hypertensive agents, anti-platelet agents or anti-coagulation.⁶ Surgical revascularization, or

more increasingly endovascular stenting, is typically pursued for those with significant acute kidney injury, in an attempt to rescue and preserve renal function, or in the management of resistant renovascular hypertension.⁷ Direct angiography should not be delayed cases when renal revascularization is anticipated to be of benefit, in order to reduce the chance of renal infarction.⁸ Nephrectomy may also be considered for patients with uncontrolled renovascular hypertension and radiological evidence of gross renal infarction or a poorly functioning kidney on a renal isotope scan. However, there remains no clear guideline or consensus as to the optimal therapy for SRAD.

For our patient, with a delayed diagnosis, established renal parenchymal loss and medically controlled hypertension, revascularization is unlikely to be feasible or of benefit at this juncture. The natural history of SRAD in this scenario is unclear. Hypertension occurs in almost all patients, and future occurrence of malignant hypertension and progressive renal failure has been described.⁹ Hence, long-term follow-up is prudent to ensure optimal blood pressure control, cardiovascular risk management and surveillance of kidney function.

REFERENCES

- [1] Béroniade V, Roy P, Froment D, Pison C. Am J Nephrol. 1987; 7(5):382-9. Primary renal artery dissection. Presentation of two cases and brief review of the literature.
- [2] Afshinnia F1, Sundaram B, Rao P, Stanley J, Bitzer M. Evaluation of characteristics, associations and clinical course of isolated spontaneous renal artery dissection. Nephrol Dial Transplant. 2013 Aug; 28(8): 2089-98
- [3] S P Stawicki, J C Rosenfeld, N Weger, E L Fields, J D Balshi Spontaneous renal artery dissection: three cases and clinical algorithms J Hum Hypertension vol 20, 710–718 (2006)
- [4] Basile C, Lisi P, Chimienti D, Antonelli M, Bruno A, Giambersio S, Zurlo MT, Petronelli S. Fibromuscular dysplasia of renal arteries presenting with bilateral renal infarction in a young man. J Nephrol. 2013 Sep-Oct; 26(5):945-8.
- [5] Alamir A, Middendorf DF, Baker P, Nahman NS Jr, Fontaine AB, Hebert LA. Renal artery dissection causing renal infarction in otherwise healthy men. Am J Kidney Dis. 1997 Dec; 30(6):851-5.
- [6] Misrai V, Peyromaure M, Poiree S. Spontaneous dissection of branch renal artery-is conservative management safe and effective? J Urol. 2006; 176(5): 2125–2129.
- [7] García-Familiar A, Ortiz-Gutiérrez F, De Blas-Bravo M, Sánchez-Abuín J, Rodríguez Sáenz de Buruaga V, Egaña-Barrenechea JM. Isolated spontaneous renal artery dissection: endovascular management. Ann Vasc Surg. 2014 May; 28(4): 1034.e5-8.
- [8] Vitiello GA, Blumberg SN, Sadek M. Endovascular Treatment of Spontaneous Renal Artery Dissection After Failure of Medical Management. Direct Vasc Endovascular Surg. 2017 Oct; 51(7):509-512.
- [9] Smith BM, Holcomb GW 3rd, Richie RE, Dean RH. Renal artery dissection. Ann Surg. 1984 Aug; 200(2): 134-46.

Citation: Tamara Wanigasekera, Rosemary Lucey, Ayanfeoluwa Obilana, Kevin O'Regan, Frank L. Ward. Spontaneous Renal Artery Dissection: A Case Report and Brief Review of the Literature. ARC Journal of Urology. 2018; 3(3):10-12. doi:dx.doi.org/10.20431/2456-060X.0303003.

Copyright: © 2018 Authors. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.