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Case Report

Renal artery stenting reversing ischemic nephropathy

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Background: Atherosclerotic renal artery stenosis (RAS) is a significant cause of renal failure, especially in patients with a solitary functioning kidney. Timely revascularization can potentially reverse ischemic nephropathy and prevent further complications like pulmonary edema and uncontrolled hypertension.

Case Presentation: A 71-year-old male with a solitary functioning kidney presented with worsening renal function, chronic hyponatremia, and recurrent flash pulmonary edema. The patient presented with a 95% ostial stenosis of the right renal artery. Percutaneous renal artery stenting was performed successfully, resulting in improved clinical status and renal function. Serum creatinine decreased from 3.07 mg/dL to 2.1 mg/dL post-procedure. The patient remained stable at a two-month follow-up.

Conclusion: Renal artery stenting in patients with ischemic nephropathy secondary to RAS can significantly improve renal function and overall clinical outcomes. Early intervention should be considered in similar cases to prevent irreversible renal damage.

Keywords: *renal artery stenosis, renal artery stenting, atherosclerosis, renal failure, ischemic nephropathy*

Introduction

Atherosclerotic renal artery stenosis is the most common cause of renal artery stenosis (RAS) in patients more than 45 years of age and is associated with ischemic nephropathy, renal failure, and secondary hypertension. Patients with solitary functioning kidney developing hemodynamically significant renal artery stenosis can result in rapidly declining renal failure and flash pulmonary edema (Garovic & Textor, 2005; Zeller et al., 2003). Indications for renal artery revascularization include symptomatic RAS with cardiac destabilization syndromes, renal function decline, malignant hypertension, asymptomatic cases with bilateral RAS or unilateral RAS in solitary kidney in an attempt to preserve renal mass.

In recent years, advancements in the techniques and technologies used for renal artery stenting have shown promising results in improving both procedural success and clinical outcomes. Studies have highlighted the use of drug-eluting stents (DES) and intravascular ultrasound (IVUS) for more accurate stent deployment, which has been associated with reduced restenosis rates and better long-term patency. For instance, Zähringer et al. demonstrated that the use of DES in renal artery stenting significantly decreased the rate of restenosis compared to bare metal stents (BMS) (Zähringer et al., 2007). Additionally, IVUS has been shown to improve stent placement precision, leading to improved renal function outcomes post-procedure (Takumi et al., 2011). These developments have allowed for more personalized patient care, especially in complex cases such as those involving solitary kidneys and significant stenosis.

Case presentation

A 71-year-old gentleman with a single functioning kidney presented with acute on chronic renal failure, deterioration of previously medically controlled hypertension, chronic hyponatremia, and a recent history of flash pulmonary edema. He had a history of coronary artery bypass grafting surgery done for triple vessel disease five years back. His blood pressure was 190/100 mmHg on four antihypertensive drugs. Initial lab investigations showed hemoglobin 9.6 g/dl, creatinine 3.07 mg/dl, sodium 122 mEq/L, and potassium 5 mEql/L. Other labs were unremarkable. His renal function had deteriorated in the last 3 weeks from a baseline creatinine of 1.9 mg/dl. The electrocardiogram showed sinus rhythm and left ventricular hypertrophy with strain. The echocardiogram showed normal biventricular function, concentric left ventricular hypertrophy, no regional wall motion abnormality, and no significant valvular lesions. MR Renal Angiogram showed a completely occluded left renal artery, a small-sized left kidney with multiple cortical cysts, and a focal high-grade stenosis of the ostial right renal artery with post-stenotic dilatation, with normal right kidney size. A renal angiogram revealed a 95% stenosis at the ostium of the right renal artery (Figure 1).

Based on the recent deterioration in renal function, uncontrolled hypertension, and history of flash pulmonary edema, we decided to proceed with the percutaneous intervention of the culprit lesion. The right renal artery was engaged with a renal double curve (RDC) 7F guide catheter, and the lesion crossed with a sion blue (ASAHI INTECC, Japan) workhorse wire. After crossing, the lesion was sequentially dilated with semicompliant balloon MINI TREK 2x12 mm @ 12 ATM (Abbott Vascular, USA) and non-compliant balloon NC TREK 3.5 x 12 mm @ 16 ATM (Abbott Vascular, USA). The RX Herculink Elite (Abbott Vascular, USA) bare metal stent 5 x 18 mm was deployed at 11 ATM, and the proximal stent edge was flared in the aorta (Figure 2). Post-stent angiogram showed good stent expansion and flow with no complications (Figure 3). He showed dramatic improvement in clinical status and renal function parameters, with a decline in serum creatinine to 2.1 mg/dl two days after the procedure. He was discharged on dual antiplatelets (aspirin and clopidogrel), statins, antihypertensives, and low-dose diuretics and remained stable on a two-month follow-up.

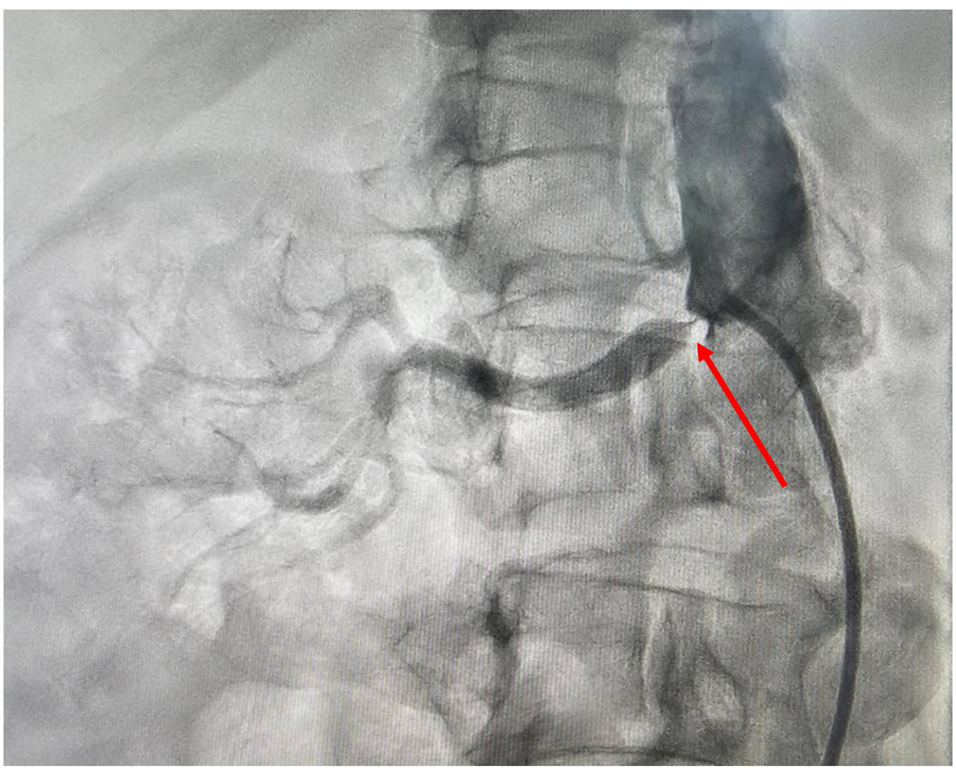

Figure 1. This figure illustrates the 95% ostial stenosis responsible for the patient's ischemic nephropathy and clinical deterioration

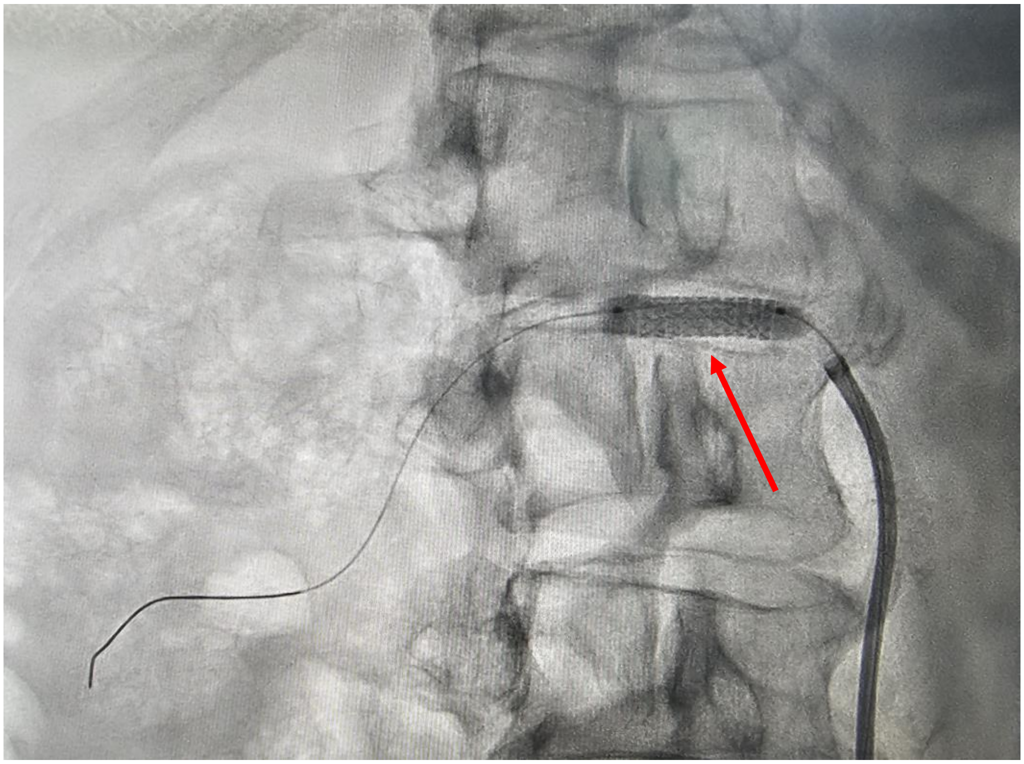

Figure 2. This figure shows the successful deployment of a bare metal stent, restoring adequate blood flow through the stenosed right renal artery

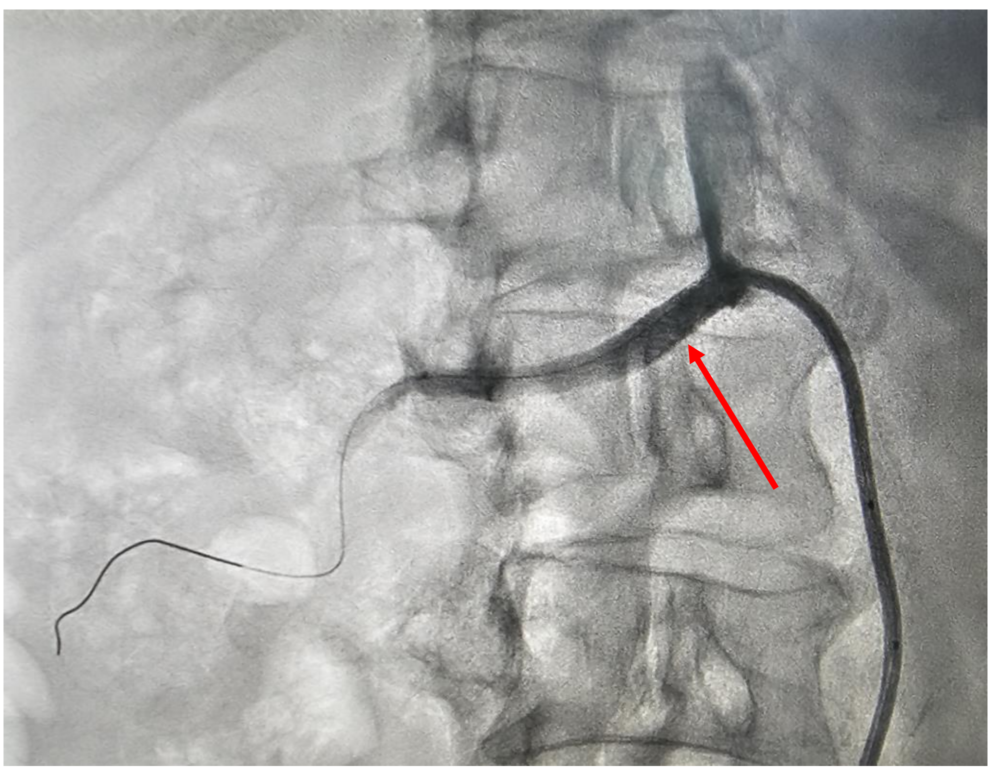

Figure 3. This post-stent angiogram shows restored flow, correlating with the patient's improved renal function

Discussion

Renal hypoperfusion is a strong stimulus for renal neurohormonal activation, resulting in renal and angiotensin II release (Agarwal et al., 1999). La Batide-Alanore et al. showed that bilateral renal artery disease or comparable conditions like unilateral renal artery stenosis with a single functioning kidney, differed from unilateral stenosis with bilateral functioning kidneys in the mechanism by which fluid overload precipitates (La Batide-Alanore et al., 2001). Bilateral RAS causes pulmonary edema secondary to volume overload. Unilateral RAS seems to offset the renal angiotensin-II pathway somehow. Following RAS, renin is released from the juxtaglomerular apparatus, causing intravascular expansion via sodium and water retention. The ensuing volume overload causes increased left atrial pressure, releasing natriuretic peptides, leading to effective natriuresis via the normal kidney. However, in bilateral RAS or unilateral RAS with a single functioning kidney, volume overload causes increased left atrial pressure and pulmonary edema as the protective mechanisms are impaired. In ischemic nephropathy, several deleterious metabolic pathways in the post-stenotic kidney have been implicated in the progression of this disease including microvascular damage, oxidative stress, inflammation, and development of fibrosis (Eirin & Lerman, 2013). The extent and severity of the damage to the post-stenotic kidney may play an important role in renal recovery and outcomes of renal angioplasty. The reversibility of these processes has not yet been fully established (Saad et al., 2013).

In patients suspected of having RAS, duplex Doppler ultrasonography and CT/MRI angiography may be used for initial evaluation (Liang et al., 2017). A peak systolic velocity (PSV) >180 cm/s has a 95% sensitivity and 90% specificity for significant RAS. When the ratio of the PSV of the stenosed renal artery to the PSV in the aorta is >3.5, DUS predicts >60% RAS with a 92% sensitivity (Chi et al., 2009). CT/MRI angiography allows localization, enumeration of the renal arteries, and characterization of the stenosis. Compared with invasive angiography, it has a sensitivity of 97% and specificity of 93% for detection of RAS (Kawashima et al., 2000; Tan et al., 2002). However, conventional angiography remains the gold standard for diagnosis. Renal artery stenosis of >80% is considered severe, 50-80% as moderate stenosis. Patients with moderate stenosis may need further assessment using a captopril renal scan, pressure gradient, or renal artery FFR (fractional flow reserve) assessment to determine functional significance. An expert panel of the American Heart Association recommended that a peak systolic gradient of at least 20 mmHg or a mean pressure gradient of 10 mmHg be used to identify candidate lesions for revascularization in symptomatic patients with RAS (Rundback et al., 2002). Renal FFR >/= 0.80 is considered normal, and <0.80 is abnormal (Mitchell et al., 2007).

Renal artery stenting may be done from the radial, brachial, or femoral approach. The ACCESS study showed access site-related bleeding complications of 2.3% for the brachial group, 2.3% for the femoral group, and 0% for the radial group (Kiemeneij et al., 1997). To avoid ostial manipulation by the guiding catheter, catheter in catheter technique (Safian & Madder, 2009) or no touch technique (Feldman et al., 1999) may be used. Despite excellent angiographic outcomes achieved with renal stenting, there is a mismatch between angiographic (>97%) and clinical (~70%) success. The plaque composition of RAS has been postulated to be responsible for some of the observed variability in clinical response in renal function after intervention. Virtual histology intravascular ultrasound (VH-IVUS) allows characterization of the percentage of necrotic core in atherosclerotic plaque, which has been correlated with worsening renal function post-intervention, likely due to microembolization of necrotic debris (Takumi et al., 2011). A study by Cooper et al. showed that the combined use of an embolic protection device (EPD) and abciximab might protect from post-procedure eGFR decline in renal artery stenting (Cooper et al., 2008). The GREAT study evaluated restenosis after stent angioplasty of atherosclerotic RAS with bare metal stent (BMS) vs drug-eluting stents (DES). It showed at 1 year follow-up clinical patency was 88.5% in the BMS and 98.1% in the DES group (Zähringer et al., 2007). Another study of renal artery stenting showed that vessels with a diameter smaller than 4.5 mm had a restenosis rate of 36%, compared with a 6.5% restenosis rate in vessels with diameters greater than 6 mm (Lederman et al., 2001). Using physiologic confirmation of RAS severity before intervention, intravascular ultrasound to maximize safe deployment diameters, and imaging modalities to identify adequate renal parenchymal reserve are some of the strategies that may improve the response rate from the 70 to >90% for patients undergoing renal stenting.

Conclusion

Renal artery stenting has demonstrated its efficacy in significantly improving renal function and clinical outcomes in patients with ischemic nephropathy secondary to renal artery stenosis, particularly in those with a solitary functioning kidney. This case underscores the critical importance of early intervention to prevent irreversible renal damage, with notable improvements in renal function, hypertension control, and prevention of recurrent pulmonary edema. Furthermore, integrating renal artery stenting into broader clinical practice offers a valuable option for mitigating the progression of chronic kidney disease and reducing cardiovascular events related to renal ischemia. Future clinical management should emphasize timely diagnosis and revascularization, which can improve patient outcomes, particularly in high-risk populations with advanced atherosclerotic disease.

Author contribution

RN and SD reported the cases and wrote the manuscript. Supervised by PR, KL Patients were under the care of PR and managed by KL, PR. All authors approved the final version of the manuscript.

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AI tool declaration

The authors declare that no AI and associated tools are used for writing scientific content in the article.

Conflict of interest

The author declares no conflict of interest. The manuscript has not been submitted for publication in other journal.

Informed consent

We have taken informed consent from the patient and also we have not disclosed the identity of the patient.

References

Agarwal, M., Lynn, K. L., Richards, A. M., & Nicholls, M. G. (1999). Hyponatremic-hypertensive syndrome with renal ischemia: An underrecognized disorder. *Hypertension, 33*(4), 1020–1024. https://doi.org/10.1161/01.HYP.33.4.1020

Chi, Y. W., White, C. J., Thornton, S., & Milani, R. V. (2009). Ultrasound velocity criteria for renal in-stent restenosis. *Journal of Vascular Surgery, 50*(1), 119–123. https://doi.org/10.1016/j.jvs.2008.12.066

Cooper, C. J., Haller, S. T., Colyer, W., Steffes, M., Burket, M. W., Thomas, W. J., Safian, R., Reddy, B., Brewster, P., Ankenbrandt, M. A., Virmani, R., Dippel, E., Rocha-Singh, K., Murphy, T. P., Kennedy, D. J., Shapiro, J. I., D'Agostino, R. D., Pencina, M. J., & Khuder, S. (2008). Embolic protection and platelet inhibition during renal artery stenting. *Circulation, 117*(21), 2752–2760. https://doi.org/10.1161/CIRCULATIONAHA.107.730259

Eirin, A., & Lerman, L. O. (2013). Darkness at the End of the Tunnel: Poststenotic Kidney Injury. *Physiology, 28*(4), 245. https://doi.org/10.1152/PHYSIOL.00010.2013

Feldman, R. L., Wargovich, T. J., & Bittl, J. A. (1999). Pearls No-Touch Technique for Reducing Aortic Wall Trauma During Renal Artery Stenting. *Catheterization and Cardiovascular Interventions : Official Journal of the Society for Cardiac Angiography & Interventions, 2*(46), 245–248. https://doi.org/https://doi.org/10.1002/(SICI)1522-726X(199902)46:2<245::AID-CCD27>3.0.CO;2-V

Garovic, V. D., & Textor, S. C. (2005). Renovascular hypertension and ischemic nephropathy. *Circulation, 112*(9), 1362–1374. https://doi.org/10.1161/CIRCULATIONAHA.104.492348

Kawashima, A., Sandler, C. M., Ernst, R. D., Tamm, E. P., Goldman, S. M., & Fishman, E. K. (2000). CT Evaluation of Renovascular Disease. *RadioGraphics, 20*(5), 1321–1340. https://doi.org/10.1148/RADIOGRAPHICS.20.5.G00SE141321

Kiemeneij, F., Laarman, G. J., Odekerken, D., Slagboom, T., & Van Der Wieken, R. (1997). A Randomized Comparison of Percutaneous Transluminal Coronary Angioplasty by the Radial, Brachial and Femoral Approaches: The Access Study. *Journal of the American College of Cardiology, 29*(6), 1269–1275. https://doi.org/10.1016/S0735-1097(97)00064-8

La Batide-Alanore, A., Azizi, M., Froissart, M., Raynaud, A., & Plouin, P. F. (2001). Split renal function outcome after renal angioplasty in patients with unilateral renal artery stenosis. *Journal of the American Society of Nephrology, 12*(6), 1235–1241. https://doi.org/10.1681/ASN.V1261235

Lederman, R. J., Mendelsohn, F. O., Santos, R., Phillips, H. R., Stack, R. S., & Crowley, J. J. (2001). Primary renal artery stenting: Characteristics and outcomes after 363 procedures. *American Heart Journal*, *142*(2), 314–323. https://doi.org/10.1067/MHJ.2001.116958

Liang, K. W., Chen, J. W., Huang, H. H., Su, C. H., Tyan, Y. S., & Tsao, T. F. (2017). The Performance of Noncontrast Magnetic Resonance Angiography in Detecting Renal Artery Stenosis as Compared with Contrast Enhanced Magnetic Resonance Angiography Using Conventional Angiography as a Reference. *Journal of Computer Assisted Tomography*, *41*(4), 619–627. https://doi.org/10.1097/RCT.0000000000000574

Mitchell, J. A., Subramanian, R., White, C. J., Soukas, P. A., Almagor, Y., Stewart, R. E., & Rosenfield, K. (2007). Predicting blood pressure improvement in hypertensive patients after renal artery stent placement. *Catheterization and Cardiovascular Interventions*, *69*(5), 685–689. https://doi.org/10.1002/CCD.21095

Rundback, J. H., Sacks, D., Kent, K. C., Cooper, C., Jones, D., Murphy, T., Rosenfield, K., White, C., Bettmann, M., Cortell, S., Puschett, J., Clair, D., & Cole, P. (2002). Guidelines for the reporting of renal artery revascularization in clinical trials. *Circulation*, *106*(12), 1572–1585. https://doi.org/10.1161/01.CIR.0000029805.87199.45

Saad, A., Herrmann, S. M. S., Crane, J., Glockner, J. F., Mckusick, M. A., Misra, S., Eirin, A., Ebrahimi, B., Lerman, L. O., & Textor, S. C. (2013). Stent revascularization restores cortical blood flow and reverses tissue hypoxia in atherosclerotic renal artery stenosis but fails to reverse inflammatory pathways or glomerular filtration rate. Circulation: *Cardiovascular Interventions*, *6*(4), 428–435. https://doi.org/10.1161/CIRCINTERVENTIONS.113.000219

Safian, R. D., & Madder, R. D. (2009). Refining the Approach to Renal Artery Revascularization. JACC: *Cardiovascular Interventions*, *2*(3), 161–174. https://doi.org/10.1016/J.JCIN.2008.10.014

Takumi, T., Mathew, V., Barsness, G. W., Kataoka, T., Rubinshtein, R., Rihal, C. S., Gulati, R., Eeckhout, E., Lennon, R. J., Lerman, L. O., & Lerman, A. (2011). The association between renal atherosclerotic plaque characteristics and renal function before and after renal artery intervention. *Mayo Clinic Proceedings*, *86*(12), 1165–1172. https://doi.org/10.4065/mcp.2011.0302

Tan, K. T., van Beek, E. J. R., Brown, P. W. G., Van Delden, O. M., Tijssen, J., & Ramsay, L. E. (2002). Magnetic Resonance Angiography for the Diagnosis of Renal Artery Stenosis: A Meta-analysis. *Clinical Radiology*, *57*(7), 617–624. https://doi.org/10.1053/CRAD.2002.0941

Zähringer, M., Sapoval, M., Pattynama, P. M. T., Rabbia, C., Vignali, C., Maleux, G., Boyer, L., Szczerbo-Trojanowska, M., Jaschke, W., Hafsahl, G., Downes, M., Bérégi, J.-P., Veeger, N. J. G. M., Stoll, H.-P., & Talen, A. (2007). Sirolimus-eluting versus bare-metal low-profile stent for renal artery treatment (GREAT Trial): angiographic follow-up after 6 months and clinical outcome up to 2 years. *Journal of Endovascular Therapy : An Official Journal of the International Society of Endovascular Specialists*, *14*(4), 460–468. https://doi.org/10.1177/152660280701400405

Zeller, T., Frank, U., Müller, C., Bürgelin, K., Sinn, L., Bestehorn, H. P., Cook-Bruns, N., & Neumann, F. J. (2003). Predictors of Improved Renal Function After Percutaneous Stent-Supported Angioplasty of Severe Atherosclerotic Ostial Renal Artery Stenosis. *Circulation*, *108*(18), 2244–2249. https://doi.org/10.1161/01.CIR.0000095786.44712.2A