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# Post-stenotic renal artery dilatation – the smoking gun of a case of renal fibromuscular dysplasia induced secondary hypertension

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## ABSTRACT

**INTRODUCTION:** Renal artery stenosis is a leading cause of secondary hypertension, with atherosclerosis and fibromuscular dysplasia as the main causes.

**CASE REPORT:** A 41-year-old male developed progressive worsening of high blood pressure over the previous 5 years, along with erythrocytosis, despite compliance with dual antihypertensive therapy. Duplex ultrasound suggested the presence of a right renal artery stenosis. Subsequent tomography angiography (CTA) failed to report a significant stenosis. However, upon imaging reappraisal, the presence of a mid-section dilatation in the right renal artery was noted. Digital subtraction angiography confirmed the dilatation between 2 focal stenoses, which were not identifiable on CTA, suggestive of fibromuscular dysplasia. A translesional pressure gradient of 107mmHg was determined. Renal artery stenting was performed with a 7x27mm covered stent with good angiographic result. Nine months after intervention, blood pressure normalized, and antihypertensive medications were withdrawn.

**CONCLUSION:** High clinical suspicion was key to adequately diagnose and manage this case of renovascular secondary hypertension.

**Keywords:** Renal artery obstruction;Fibromuscular dysplasia; Renovascular hypertension, Renal angioplasty.

## INTRODUCTION

Secondary hypertension is defined as hypertension attributable to an identifiable cause and accounts for 5–10% of cases of systemic hypertension.[1] Renal artery stenosis (RAS) is defined as a >50% reduction in the lumen of at least one renal artery or its branches, resulting in impaired renal perfusion. RAS is a leading cause of secondary hypertension, with prevalence of 1–8% among hypertensive patients, and may affect 25–35% of patients with generalized atherosclerosis.1 Fibromuscular dysplasia of the renal artery (RA) is a common cause of RAS that should be considered in children and young adults, whereas atherosclerotic RAS is more prevalent in older adults.[2] RAS with impaired renal perfusion leads to activation of the renin-aldosterone-angiotensin system, which increases sodium and water retention and systemic vasoconstriction, resulting in a rise in blood pressure (BP). Typically, renovascular hypertension

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leads to ischemic nephropathy, presenting clinically as severe hypertension that is refractory to medical management, because of angiotensin-II-mediated stimuli. Depending on the presence and functional status of the contralateral kidney, insufficient sodium and water excretion may occur, leading to volume overload, hypertensive crisis, flash pulmonary edema, congestive heart failure, and stroke.3 We present the case of an adult with cardiovascular risk factors who developed secondary hypertension and erythrocytosis insidiously due to RAS of unclear etiology.

## CASE REPORT

A 41-year-old male with dyslipidemia, hyperuricemia, and smoking habits (5 pack-years) was diagnosed with arterial hypertension five years ago. He was medicated with 5 mg of amlodipine and 5 mg of ramipril per day.

His family history revealed a grandmother who underwent a nephrectomy for renal cell cancer and who developed end-stage renal failure requiring peritoneal dialysis, and a father with hypertension requiring medication.

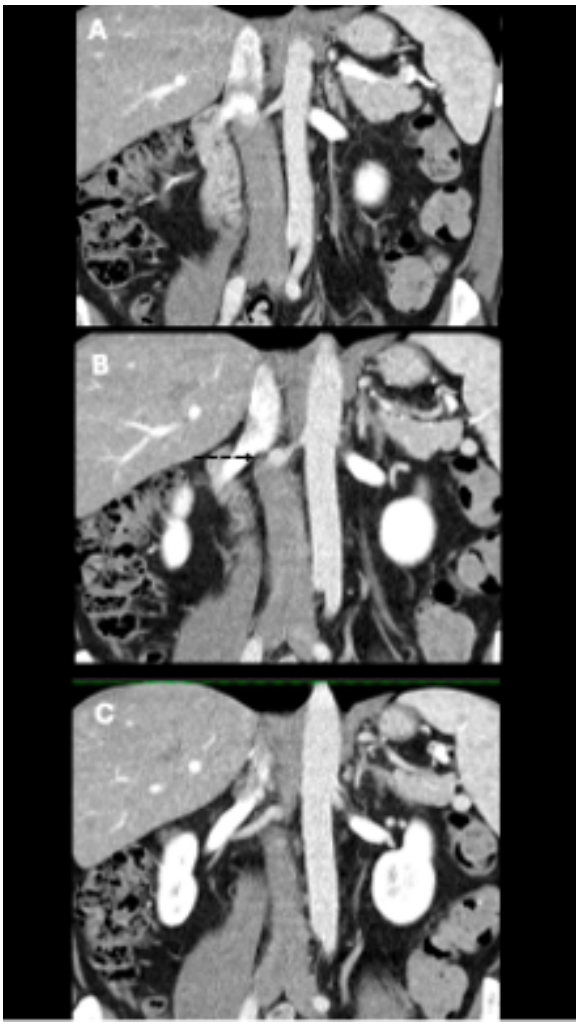
On vascular assessment, all peripheral arterial pulses were present. No limb BP asymmetry or carotid or abdominal bruits were found.

Over the past 3 years, the patient presented with a progressive increase in BP (systolic BP of 160 mmHg) despite strict adherence to antihypertensive medication and dietary sodium restriction. No hypertensive crisis or target-organ lesions were reported. No cardiac failure was detected on echocardiography. Notably, he also developed progressive, asymptomatic erythrocytosis, with hemoglobin levels reaching 19.1 g/dL, requiring three sessions of phlebotomy.

Due to his atypical presentation, secondary hypertension was suspected and screened. Laboratory blood tests revealed increased serum and urinary aldosterone levels (370.5 pg/mL and 19.66 mcg/24h, respectively) with normal plasma renin activity (3.26 ng/mL/h). The aldosterone (ng/dL)/renin (ng/mL/h) ratio was normal (11.35). Serum creatinine fluctuated from 0.91 to 1.37 mg/dL over the past three years. LDL cholesterol was 126 mg/dL, HDL cholesterol was 46 mg/dL, and total cholesterol was 187 mg/dL. Triglycerides were 166 mg/dL. Erythrocyte sedimentation rate was not determined, but C-reactive protein was 0.2 mg/dL 1 year prior. Serum cortisol, 24-hour urine metanephrines, TSH, T3, and T4 were normal. Genetic assessment for polycythemia vera was negative for JAK2 mutations. Erythropoietin (Epo) was not determined.

A renal duplex ultrasound (DUS) performed 2 months before the vascular surgery consultation identified a discretely decreased right kidney size (bipolar diameter of 82 mm), with normal parenchymal thickness and increased cortical echogenicity. It also reported a resistance index of 0.48. The maximum peak systolic velocity recorded was 76 cm/s at the right renal artery (RRA) origin, while the renal-to-aorta ratio (RAR) was not reported. However, RRA stenosis could not be excluded because the midsection of the artery could not be evaluated. No waveform descriptions or DUS images were available for assessment. Given the inconclusive DUS report, a contrast-enhanced abdominal and pelvic computerized tomography (CTA) was ordered. It corroborated renal size asymmetry, but no evidence of RAS was reported. Upon CTA imaging reappraisal during the vascular consultation, although a stenosis could not be identified, a focal dilatation of the midportion of the RRA (maximum diameter of 9 mm) was noticed (Figure 1 A-C), raising suspicion of a hemodynamically significant stenosis. Invasive angiography was ordered for clarification.

**Figure 1.** Coronal reconstruction of preoperative CTA.



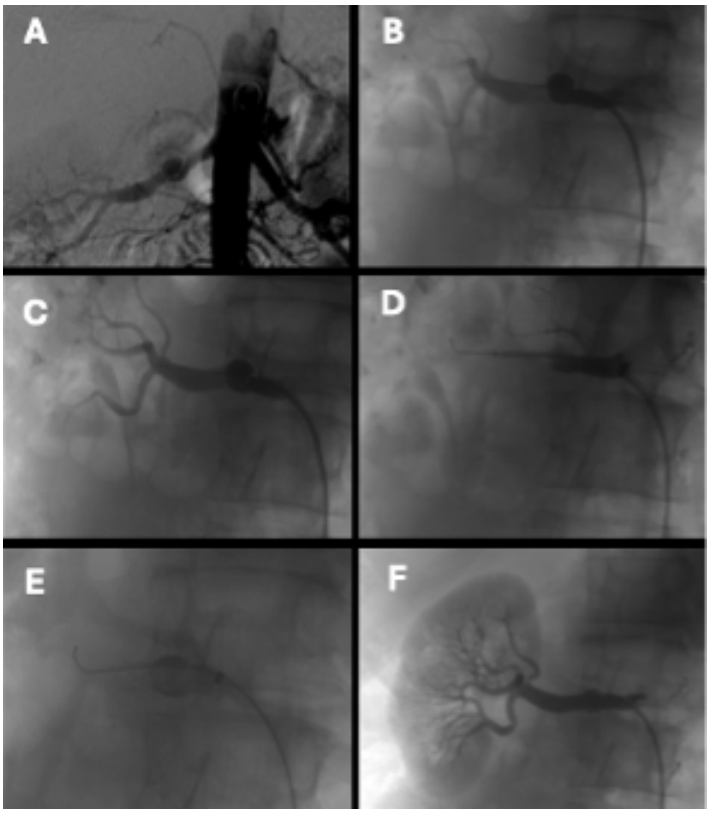

A dilatation of the midsection of the RRA is identifiable (dashed arrow, Figure 1B).

Arteriography was performed via a left femoral approach using a 5Fr Cobra catheter to cannulate the RRA. Wire advancement was possible only with a 0.018'' guidewire (Abbott Command 18) after failure to advance a 0.035'' hydrophilic guidewire (Terumo Glidewire) due to mid-RRA stenosis. During wire progression, a second stenosis was identified immediately after the arterial dilatation. Arteriography imaging showed a significant stenosis followed by post-stenotic RRA ectasia (Figure 2A-D). A translesional pressure gradient of 107mmHg (164mmHg at the RRA ostium, 57 mmHg post-stenosis) was recorded.

RRA primary stent angioplasty was performed without filter protection using a 7x27mm balloon-expandable covered stent (Bentley Begraft Peripheral), with brisk flow appreciated on control angiography (Figure E and F). The patient was discharged the next day uneventfully on dual antiplatelet therapy (100mg of aspirin and 75 mg of clopidogrel per day for 3 months, with aspirin continued long-term), while statin therapy was refused by the patient.

A postoperative renal DUS was performed 6 months later, confirming a normal right kidney RI of 0.55 and no arterial waveform abnormalities. Nine months after the intervention, due to progressive lowering of blood pressure, the patient has discontinued all antihypertensive medication. The patient was scheduled for annual DUS assessment.

**Figure 2.** Angiography and right renal stenting.




A – Non-selective aortography demonstrating presence of the midsection dilatation of the RRA. B – Selective angiography from 7Fr sheath with backflow into the abdominal aorta, prior to right kidney arteriogram completion. C- Right kidney arteriography. D - Distal occlusion of the RRA by stent mounted on delivery balloon catheter. E – Implanted Bentley peripheral Begraft 7x27mm. Notice residual contrast capture within the renal artery dilatation. F – Final RRA angiography demonstrating stent patency and perm**eabi**lity right kidney arterial vascularization.

## DISCUSSION

This case report underscores that a high level of clinical suspicion and careful imaging reappraisal were essential to identify RAS and ensure its adequate management.

Secondary hypertension affects 5-25% of hypertensive adults.[4] While screening is mandatory in adults with resistant hypertension, it is also recommended when clinical suspicion is present.[4] In our case, given the late and insidious onset of hypertension in a 41-year-old smoker, initial suspicion and screening for secondary hypertension might not be recommended.[1,4] Nevertheless, the progressive worsening of BP despite continuous adherence to dietary restrictions could suggest an underlying secondary hypertension superimposed on essential hypertension.

Revascularization is recommended for severe RAS (>60% stenosis, translesional gradient >30 mmHg, RAR > 3.5:1) when associated with resistant hypertension (BP above goal despite treatment with 3 or more antihypertensive drugs of different classes with complementary mechanisms, including a diuretic at maximally tolerated doses), rapidly deteriorating renal function, recurrent hypertensive crisis, or flash pulmonary edema.[4,5] Despite our patient not fitting any of these clinical patterns, the progressive BP increase associated with elevated red blood cell (RBC) counts (after polycythemia vera was excluded) prompted further invasive investigation. RAS is a known cause of acquired secondary erythrocytosis through impaired renal perfusion and local renal hypoxia, which may develop with or without elevated Epo.[6-9] This was the main finding, along with progressive worsening of BP, which led us to order invasive imaging despite inconclusive previous duplex and CT scans. Renal revascularization has previously been associated with improved RBC counts in two separate case reports.[8,9] However, in both cases, JAK-2 mutations were present, and concomitant treatment with hydroxyurea was commenced, which prevents firm conclusions.

In our patient, BP control improved after renal artery stenting. Several single-center and individual series have reported BP improvement in 50-80% of cases, with improved or stabilized renal function in 70-85% of patients.[10,11] However, these benefits have not been reproduced in several randomized controlled trials (RCTs) (EMMA, DRASTIC, STAR, ASTRAL, CORAL), either individually or when pooled, except for refractory hypertension within 2 years of follow-up (OR 0.09, 95% CI 0.01-0.7).[12-18] In consequence, for most patients with atherosclerotic RAS with controlled hypertension and stable renal function on medical treatment, renal artery revascularization does not provide additional benefit. However, because of heterogeneous and inadequate inclusion criteria, imprecise RAS severity assessment, and inappropriate study endpoint selection in the aforementioned RCTs, patient subgroups that might benefit from intervention remain undetermined.

DUS remains the first-line imaging modality for identifying RAS, providing direct criteria such as increased peak systolic velocity (PSV) at the stenosis site (> 180-285 cm/s for 50% stenosis) or a reno-aortic PSV ratio >3.5 for >60% stenosis.[5] Indirect criteria include renal resistance index asymmetry, waveform shape, flow acceleration, and delayed acceleration time (rise time >0.07s, tardus parvus waveform). CTA and magnetic resonance angiography remain the preferred noninvasive imaging modalities, with reported sensitivities of 64-100% and 70-97%, and specificities ranging from 62-98% and 90%, respectively.[5] In this case, RAS was not initially detected. However, on reappraisal, midportion RRA ectasia was noted, suggesting significant arterial stenosis.

Hemodynamically significant stenoses induce arterial dilatation due to flow turbulence, low-pressure gradients, and flow recirculation patterns, along with increased nitric oxide release, producing compensatory arterial vasodilatation.[19-21]

The etiology of the RRA stenosis remains unclear. Although our patient's age, gender, and cardiovascular risk factors support an atherosclerotic cause, the focal morphology, mid-arterial location, and the presence of two distinct stenoses separated by a short focal arterial dilatation are consistent with fibromuscular dysplasia (FMD).[22,23] Indeed, although FMD is considered rare, with prevalence estimates of approximately 0.02-0.08%, recent data suggest it is more common, with one study reporting a prevalence of 4% among a potential kidney donor population.[24-26]

Primary covered stent implantation was preferred to exclude arterial dilatation and to treat both stenoses simultaneously, given the uncertainty of the disease etiology and the subsequent evolution of the dilatation. Although renal artery stenting is recommended over plain balloon angioplasty (POBA), especially for atherosclerotic stenosis, POBA is sufficient for FMD stenoses; however, data supporting this recommendation are scarce.[5] In a study by Dorros et al comparing renal artery POBA to stenting, stenting was associated with improved technical success and pressure gradient reduction.[27] A single RCT by Ven et al has been published on this subject.[28] Similarly, in a group of 84 patients randomized to renal artery stenting or POBA, procedure success and long-term patency were favored in the stent group, while 29% of the patients in the POBA group crossed over to the stent group. Although 40% of patients initially avoided stent placement, 45% ultimately required a second procedure with stenting, whereas only 12% in the primary stenting group required a second procedure. A meta-analysis comparing primary stenting to POBA for atherosclerotic RAS confirmed improved procedural success for stenting vs POBA (98% vs 77%, $p < 0.001$) and lower restenosis rates (17 vs 26%, respectively, $p < 0.001$).[19]

While renal artery stenting improves durability, data supporting covered stents over bare-metal stents are practically nonexistent. In the single-arm ARTISAN study, 68 patients received covered stents (iCAST RX), with 70% primary patency at 9 months and a clinically driven revascularization rate of 7.3% at 36 months.[30] Although limited by its small, single-arm design, these results are comparable to the stenting arm of the RCT from van de Ven et al.[28] While a prospective RCT (Covered vs Bare Metal Stents for Atherosclerotic Renal Artery Stenosis [COMBAT] – NCT07566065) is currently awaited, inference from data in the mesenteric territory may suggest superiority of covered over bare-metal stents.[31] Given the complex lesion (double stenosis with intermediate ectasia), we chose a covered-stent strategy despite uncertain long-term benefit.

Renal function deterioration after renal artery revascularization has been attributed to iodinated contrast toxicity and arterial embolization.[32,33] Thus, embolic protection devices (EPDs) may be considered during renal artery revascularization. Two methods have been used in renal artery revascularization. Distal balloon occlusion with the Guardwire® Temporary Occlusion and Aspiration System (Medtronic, Minneapolis, MN, USA) has been associated with favorable outcomes in several single-arm retrospective studies, with most patients having visible debris recovered by aspiration.[33-35] However, no randomized controlled studies have been published using this device. Alternatively, filters are more commonly used and effectively prevent debris embolization.[36] In the only RCT published on EPD use in renal artery revascularization, the Angioguard™ (Cordis) filter (RESIST trial) yielded equivocal results, suggesting that combining filter protection with the platelet glycoprotein IIb/IIIa inhibitor abciximab may confer additional benefit in 1-month post-interventional renal function.[37] In our patient, given the uncertain pathophysiology of the stenosis, the absence of intraluminal debris on preoperative CT imaging, and the choice of primary stenting with a covered stent, a low risk of embolization was anticipated, and thus embolic protection was waived.

In conclusion, in our case presenting with erythrocytosis, progressive worsening of BP, and nonspecific imaging findings, a high clinical suspicion followed by invasive arteriography were key to enabling detection and adequate management of this case of secondary hypertension of unclear etiology.

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## REFERENCES

**1.** Rimoldi SF, Scherrer U, Messerli FH. Secondary arterial hypertension: when, who, and how to screen? Eur Heart J. 2014;35:1245-54.

**2.** Textor SC, Lerman L. Renovascular hypertension and ischemic nephropathy. Am J Hypertens. 2010;23:1159-69.

**3.** Gaspar MA. Renovascular hypertension: The current approach. Rev Port Cardiol. 2019;38:869-71.

**4.** Writing Committee Members*; Jones DW, Ferdinand KC, Taler SJ, Johnson HM, Shimbo D, et al. 2025 AHA/ACC/AANP/AAPA/ABC/ACCP/ACPM/AGS/AMA/ASPC/NMA/PCNA/SGIM Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Hypertension. 2025;82:e212-e316.

**5.** Koelemay MJ, Geelkerken RH, Kärkkäinen J, Leone N, Antoniou GA, de Bruin JL, et al. European Society for Vascular Surgery (ESVS) 2025 Clinical Practice Guidelines on the Management of Diseases of the Mesenteric and Renal Arteries and Veins. Eur J Vasc Endovasc Surg. 2025;70:153-218.

**6.** Aoun M, Jadoul M, Anders HJ. Erythrocytosis and CKD: A Review. Am J Kidney Dis. 2024;84:495-506.

**7.** Bacon BR, Rothman SA, Ricanati ES, Rashad FA. Renal artery stenosis with erythrocytosis after renal transplantation. Arch Intern Med. 1980;140:1206-11.

**8.** Bhadauria D, Sharma RK, Kaul A, Prasad N, Gupta A, Gurjar D, et al. A rare clinical syndrome of refractory secondary hypertension, renal artery stenosis and erythrocytosis. NDT Plus. 2011;4:175-7.

**9.** Gavriilaki E, Sampanis N, Kavlakoudis C, Papaioannou G, Vasileiou S. An exceptional case of renal artery restenosis in a patient with polycythaemia vera. Blood Coagul Fibrinolysis. 2014:25:904-6.

**10.** Weinberg I, Keyes MJ, Giri J, Rogers KR, Olin JW, White CJ, et al. Blood pressure response to renal artery stenting in 901 patients from five prospective multicenter FDA-approved trials. Catheter Cardiovasc Interv. 2014;83:603-9.

**11.** Moutinho M, Pedro LM, Fernandes RFE, Silva D, Fernandes JFE. Is stenting for atherosclerotic renal stenosis an effective technique? Rev Port Cardiol. 2019;38:859-67.

**12.** Plouin PF, Chatellier G, Darné B, Raynaud A. Blood pressure outcome of angioplasty in atherosclerotic renal artery stenosis: a randomized trial. Essai Multicentrique Medicaments vs Angioplastie (EMMA) Study Group. Hypertension. 1998;31:823-9.

**13.** van Jaarsveld B, Krijnen P, Bartelink A, Dees A, Derkx F, Man in't Veld A, et al. The Dutch Renal Artery Stenosis Intervention Cooperative (DRASTIC) Study: rationale, design and inclusion data. J Hypertens Suppl. 1998,16:S21-7

**14.** Bax L, Woittiez AJ, Kouwenberg HJ, Mali WP, Buskens E, Beek FJ, et al. Stent placement in patients with atherosclerotic renal artery stenosis and impaired renal function: a randomized trial. Ann Intern Med. 2009;150:840-8,

**15.** ASTRAL Investigators; Wheatley K, Ives N, Gray R, Kalra PA, Moss JG, et al. Revascularization versus medical therapy for renal-artery stenosis. N Engl J Med. 2009;361:1953-62.

**16.** Cooper CJ, Murphy TP, Cutlip DE, Jamerson K, Henrich W, Reid DM, et al. Stenting and medical therapy for atherosclerotic renal-artery stenosis. N Engl J Med. 2014;370:13-22.

**17.** Li Y, Cui WH, Wang J, Chen X, Zhang C, Zhu LZ, et al. Percutaneous revascularization for atherosclerotic renal artery stenosis: a meta-analysis. Eur Rev Med Pharmacol Sci. 2022;26:7007-14.

**18.** Chen Y, Pan H, Luo G, Li P, Dai X. Use of percutaneous transluminal renal angioplasty in atherosclerotic renal artery stenosis: a systematic review and meta-analysis. J Int Med Res. 2021;49:300060520983585.

**19.** Holman E. The obscure physiology of poststenotic dilatation; its relation to the development of aneurysms. J Thorac Surg. 1954;28:109-33.

**20.** Crotty TP. Poststenotic dilatation in arteries and the role of turbulence. Med Hypotheses. 1994;42:367-70.

**21.** Calvo WJ, Hajduczok G, Russell JA, Diamond SL. Inhibition of nitric oxide but not prostacyclin prevents poststenotic dilatation in rabbit femoral artery. Circulation. 1999;99:1069-76.

**22.** Gong X, Liu J, Yao D, Huang R. Successful treatment of focal renal artery fibromuscular dysplasia by balloon dilatation demonstrated via fractional flow reserve. Clin Med (Lond). 2023;23:625-29.

**23.** Savard S, Steichen O, Azarine A, Azizi M, Jeunemaitre X, Plouin PF. Association between 2 angiographic subtypes of renal artery fibromuscular dysplasia and clinical characteristics. Circulation. 2012;126:3062-9.

**24.** Plouin PF, Perdu J, La Batide-Alanore A, Boutouyrie P, Gimenez-Roqueplo AP, Jeunemaitre X. Fibromuscular dysplasia. Orphanet J Rare Dis. 2007 Jun 7;2:28.

**25.** McKenzie GA, Oderich GS, Kawashima A, Misra S. Renal artery fibromuscular dysplasia in 2,640 renal donor subjects: a CT angiography analysis. J Vasc Interv Radiol. 2013;24:1477–80.

**26.** Hendricks NJ, Matsumoto AH, Angle JF, Baheti A, Sabri SS, Park AW, et al. Is fibromuscular dysplasia underdiagnosed? A comparison of the prevalence of FMD seen in CORAL trial participants versus a single institution population of renal donor candidates. Vasc Med. 2014;19:363–67.

**27.** Dorros G, Prince C, Mathiak L. Stenting of a renal artery stenosis achieves better relief of the obstructive lesion than balloon angioplasty. Cathet Cardiovasc Diagn. 1993;29:191-8.

**28.** van de Ven PJ, Kaatee R, Beutler JJ, et al. Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: a randomized trial. Lancet. 1999;353:282-6.

**29.** Leertouwer TC, Gussenhoven EJ, Bosch JL, et al. Stent placement for renal artery stenosis: where do we stand? A meta-analysis. Radiology. 2000;216:78-85.

**30.** Drachman DE, Metzger DC, Jain A, Sachar R, El-Sayed Abbas A, Rosenfield K, Ansel GM. De Novo Atherosclerotic Renal Artery Stenosis Covered Stent Treatment for Resistant Hypertension (ARTISAN) Results. J Soc Cardiovasc Angiogr Interv. 2024;3:102400.

**31.** Oderich GS, Erdoes LS, Lesar C, Mendes BC, Gloviczki P, Cha S, et al. Comparison of covered stents versus bare metal stents for treatment of chronic atherosclerotic mesenteric arterial disease. J Vasc Surg. 2013;58:1316-23.

**32.** Iannone LA, Underwood PL, Nath A, Tannenbaum MA, Ghali MG, Clevenger LD. Effect of primary balloon expandable renal artery stents on long-term patency, renal function, and blood pressure in hypertensive and renal insufficient patients with renal artery stenosis. Cathet Cardiovasc Diagn. 1996;37:243-50.

**33.** Edwards MS, Corriere MA, Craven TE, Pan XM, Rapp JH, Pearce JD, et al. Atheroembolism during percutaneous renal artery revascularization. J Vasc Surg. 2007;46:55-61.

**34.** Edwards MS, Craven BL, Stafford J, Craven TE, Sauve KJ, Ayerdi J, et al. Distal embolic protection during renal artery angioplasty and stenting. J Vasc Surg. 2006;44:128-35.

**35.** Henry M, Klonaris C, Henry I, Tzetanov K, Le Borgne E, Foliguet B, et al. Protected renal stenting with the PercuSurge GuardWire device: a pilot study. J Endovasc Ther. 2001;8:227-37.

**36.** Holden A, Hill A, Jaff MR, Pilmore H. Renal artery stent revascularization with embolic protection in patients with ischemic nephropathy. Kidney Int. 2006;70:948-55.

**37.** Cooper CJ, Haller ST, Colyer W, Steffes M, Burket MW, et al. Embolic protection and platelet inhibition during renal artery stenting. Circulation. 2008;117:2752-60.