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BRIEF ARTICLE

Response of blood pressure after percutaneous transluminal renal artery angioplasty and stenting

Jayesh S Prajapati, Sharad R Jain, Hasit Joshi, Shaurin Shah, Kamal Sharma, Sibasis Sahoo, Kapil Virparia, Ashok Thakkar

Jayesh S Prajapati, Sharad R Jain, Hasit Joshi, Shaurin Shah, Kamal Sharma, Sibasis Sahoo, Kapil Virparia, Department of Cardiology, UN Mehta Institute of Cardiology and Research Centre, Ahmedabad 380016, Gujarat, India
Ashok Thakkar, Senior Clinical Trial Manager, Sahajanand, Medical Tech. Pvt. Ltd., Surat 395004, India
Author contributions: Prajapati JS, Jain SR, Joshi H, Shah S, Sharma K, Sahoo S and Virparia K performed the research; Thakkar A designed the research and wrote the paper.
Correspondence to: Dr. Jayesh S Prajapati, MD, DM, Associate Professor of Cardiology, Department of Cardiology, UN Mehta Institute of Cardiology and Research Centre, BJ Medical College and Civil Hospital Campus, Asarwa, Ahmedabad 380016, Gujarat, India. drjsprajapati@yahoo.co.in
Telephone: +91-79-26464343 Fax: +91-79-22682092
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Abstract

AIM: To evaluate the short and intermediate term outcome of percutaneous transluminal renal artery angioplasty (PTRA) and stenting particularly on blood pressure (BP) control and renal function and to evaluate predictors of poor BP response after successful PTRA and stenting.

METHODS: We conducted a prospective analysis of all patients who underwent PTRA and stenting in our institute between August 2010 to September 2012. A total number of 86 patients were underwent PTRA and renal stenting. Selective angiography was done to confirm at least 70% angiographic stenosis. The predilatation done except few cases with critical stenosis, direct stenting was done in the rest of cases. All patients received aspirin 325 mg orally, and clopidogrel 300 mg orally within 24 h before the procedure. Heparin was used as the procedural anticoagulant agent. Optimal results with TIMI-III flow obtained in all cases. Follow-ing stent placement, aspirin 150 mg orally once daily was continued for a minimum of 12 mo and clopidogrel 75 mg orally once daily for at least 4 wk. The clinical, radiological, electrocardiography, echocardiography and treatment data of all patients were recorded. The BP measurement, serum creatinine and glomerular filtration rate (GFR) were recorded before the procedure and 1 and 6 mo after PTRA.

RESULTS: A total of 86 patients were included in the study. The mean age of study population was 55.87 ± 11.85 years old and 67 (77.9%) of patients were male. There was a significant reduction in both systolic and diastolic BP at 1 mo after the procedure: 170.15 ± 20.10 mmHg vs 146.60 ± 17.32 mmHg and 98.38 ± 10.55 mmHg vs 89.88 ± 9.22 mmHg respectively (P = 0.0000). The reduction in BP was constant throughout the follow-up period and was evident 6 mo after the procedure: 144.23 ± 18.19 and 88.26 ± 9.79 mmHg respectively (P = 0.0000). However, no improvement in renal function was observed at any time during the follow-up period. After multivariate analysis, we found male sex, low GFR (< 60 mL/min) and higher baseline mean BP as a poor predictors of successful outcome on BP response after PTRA and stenting.

CONCLUSION: The PTRA and stenting can be considered as an effective therapeutic intervention for improving BP control with minimal effect on renal function. The male sex, higher baseline BP and low GFR are associated with poor BP response after successful PTRA and stenting.

Core tip: To evaluate the short and intermediate term

outcome of percutaneous transluminal renal artery angioplasty (PTRA) and stenting particularly on blood pressure (BP) control and renal function and to evaluate predictors of poor BP response after successful PTRA and stenting. The PTRA and stenting can be considered as an effective therapeutic intervention for improving BP control with minimal effect on renal function. The male sex, higher baseline BP and low glomerular filtration rate are associated with poor BP response after successful PTRA and stenting.

INTRODUCTION

Renovascular hypertension occurs in 1% to 5% of all patients with hypertension. Renovascular hypertension is the most common form of secondary hypertension. Renal artery stenosis (RAS) is caused often by atheromatous plaques (80% of the cases over 40 years), but can also be due to fibromuscular dysplasia (10% of the cases and more often in young patients), arteritis (Takayasu's disease), neurofibromatosis and post radiation injury[1-4]. It can also occur in post renal transplant patients or after a renal bypass graft[4].

RAS is associated with increased cardiovascular events and mortality. Its prevalence varies from 7% in individuals over 65 years of age to 20%-30% in high risk group of patients. It may affect up to 30% of patients with coronary artery disease and nearly 50% of those with significant peripheral vascular disease (PVD)[5,3-7]. Atherosclerotic RAS is a progressive disease associated with loss of renal mass over time, despite control of hypertension. Progression of RAS to complete occlusion is more likely with more severe (> 60%) lesions and may occur at a rate of up to 20%/year[4,8-10].

Atherosclerotic RAS is an important cause of renal insufficiency, refractory hypertension, and cardiac destabilization syndromes (unstable angina and flash pulmonary edema)[11,12]. Unilateral RAS manifests clinically as a vasoconstrictor-mediated hypertension, whereas bilateral RAS causes hypertension caused by volume overload. Up to 20% of patients older than 50 years of age requiring renal dialysis have atherosclerotic RAS (ischemic nephropathy) as the cause of their renal failure. The treatment of RAS includes medical therapy, balloon angioplasty and surgery. Surgery has been replaced by percutaneous transluminal renal artery angioplasty (PTRA) and stenting and remains at high risk with a 2%-7% perioperative mortality rate, a 17%-31% morbidity, deterioration rate in renal function in 11%-31% of patients

and reocclusion and restenosis in 5%-18%. Indications for surgery are limited and include failed percutaneous approach, hostile aorta, infra-renal total occlusion and in association with aortic surgery[4,13-15].

The PTRA technique has become the cornerstone for treatment of RAS and is now the first line treatment to be proposed. Balloon angioplasty alone was first proposed but several series reported the successful use of endovascular stents for treating suboptimal angioplasty results and as a primary intervention for atherosclerotic lesions and particularly ostial lesions with better immediate and long-term results than with balloon angioplasty alone[16-21]. Despite many reports of clinical success in selected and carefully chosen patient groups, the enthusiasm for widespread treatment of mild or moderate renovascular disease has waned. Recent published data from the Angioplasty and Stenting for Renal Artery Lesions (ASTRAL) trial, in which patients were randomized to revascularization *vs* continued medical therapy alone, did not show a clear benefit of renal revascularization, although its design and conclusions have been criticized[22]. We designed this study to evaluate the short and intermediate term outcome of PTRA and stenting particularly on blood pressure (BP) control and renal function and to evaluate predictors of poor BP response after successful PTRA and stenting.

MATERIALS AND METHODS

Study population

This study was carried out in the Department of Cardiology, UN Mehta Institute of Cardiology and Research, from August 2010 to September 2012. This institute is tertiary care center situated in Ahmedabad, Gujarat, India. A total number of 86 patients were underwent PTRA and renal stenting with following inclusion criteria: (1) significant renal artery stenosis (70% or more stenosis); (2) onset of hypertension before 30 years and after 55 years; (3) exacerbation of previously well controlled hypertension; (4) malignant hypertension and Refractory hypertension; (5) azotemia shortly after institution of therapy with ACE inhibitors or ARB blockers; (6) hypertension and atrophic kidney or discrepancy in kidney size (> 1.5 cm); (7) hypertension and recurrent episodes of acute pulmonary edema or unexplained heart failure; (8) hypertension and systolic-diastolic abdominal bruit that laterlise to one side; and (9) hypertension and progressive unexplained azotemia. The exclusion criteria were: (1) serum creatine value > 3 mg/dL; (2) small kidney; and (3) total renal artery occlusion.

Informed written consent was obtained from all patients before treatment. This study conducted in accordance with the International Conference on Harmonization guidelines Good Clinical Practices, Declaration of Helsinki, and medical ethics committee requirements.

All patients' systolic BP, diastolic BP, serum creatinine and GFR were measured at baseline, 1 mo and 6 mo respectively. The BP was measured in supine position in

Table 1   Baseline characteristics of study population: Clinical, laboratory and imaging data *n* (%)

Variables	n = 86
Male gender	67 (77.9)
Age (yr)	55.87 ± 11.85
Background diseases	
Stage-1 (malignant) hypertension	14 (16.3)
Stage-2 hypertension	65 (75.6)
Diabetes mellitus	34 (39.5)
Smoking	61 (70.9)
Clinical features of left ventricular dysfunction	23 (26.7)
Left ventricular hypertrophy	29 (33.7)
Coronary artery disease	72 (83.7)
Blood pressure	
Systolic (mmHg)	170.15 ± 20.10
Diastolic (mmHg)	98.38 ± 10.55
Antihypertensive drugs (*n*)	3.07 ± 0.69
Indication criteria	
Hypertension resistant to standard medication	71 (82.6)
Renal bruit	53 (61.6)
Serum creatinine (mg/dL)	20 (23.3)
Stenosis	
Bilateral	23 (26.7)
Coronary angiography	
Single vessel disease	25 (29.1)
Double vessel disease	14 (16.3)
Triple vessel disease	28 (32.6)
Normal vessel	19 (22.1)

Values are presented as percentage (%) and mean ± SD.

Table 2   Blood pressure, antihypertensive medication, serum creatinine and glomerular filtration rate initial *vs* follow-up measurements

Time of follow-up	mean ± SD	P value
Systolic blood pressure (mmHg)	170.15 ± 20.10	< 0.0001
Baseline		
1 mo	146.60 ± 17.32	
6 mo	144.23 ± 18.19	
Diastolic blood pressure (mmHg)	98.38 ± 10.55	< 0.0001
Baseline		
1 mo	89.88 ± 9.22	
6 mo	88.26 ± 9.79	
Antihypertensive drugs (*n*)	3.07 ± 0.69	< 0.0001
Baseline		
1 mo	2.37 ± 0.84	
6 mo	2.25 ± 0.94	
Serum creatinine (mg/dL)	1.21 ± 0.66	0.964
Baseline		
48 h	1.29 ± 0.88	
1 mo	1.33 ± 1.27	
6 mo	1.21 ± 0.79	
GFR estimation (mL/min)	65.71 ± 25.20	0.546
Baseline		
6 mo	66.68 ± 25.03	

Values are presented as mean ± SD, *P* value compares baseline to 6 mo. GFR: Glomerular filtration rate.

both upper limbs and lower limbs with mercury manometer with standard cuff size after adequate rest. Patients were not allowed to have tea, coffee, smoking and alcohol 1 h prior to procedure. Patients were allowed to continue their antihypertensive medicines. Patients were on primarily b blocker, diuretics, ace inhibitors/ARB or calcium channel blockers.

Procedure

All patients who underwent PTRA and stenting received anticoagulation as per hospital protocol. Selective angiography was done to confirm at least 70% angiographic stenosis. PTRA was performed with either 6/7 F RDC or JR 3.5 guiding catheter and work hoarse guidewire. The predilatation done except few cases with critical stenosis, direct stenting was done in the rest of cases. Post dilation was done if required. The study included bare metal stent (BMS) of 12, 15, and 18 mm lengths with diameters ranging from 4 to 7 mm.

All patients received aspirin 325 mg orally, and clopidogrel 300 mg orally within 24 h before the procedure. Heparin was used as the procedural anticoagulant agent. Optimal results with TIMI-Ⅲ flow obtained in all cases. Following stent placement, aspirin 150 mg orally once daily was continued for a minimum of 12 mo and clopidogrel 75 mg orally once daily for at least 4 wk.

Statistical analysis

All collected data entered into the "IBM SPSS STAISTICS version 20". The quantitative data expressed as mean and standard deviation (SD) where qualitative data expressed in percentage (%). The independent and dependent student's *t*-test have been used to carry out significant changes in paired and non-paired quantitative data. Also, χ^2 and Fisher exact test have been used to carry out significant change in qualitative data. The *P* value < 0.05 consider as a statistically significant. All statistically significant variables taken for univariate binary logistic regression and for univariate significant variables entered into multiple step wise logistic regression for further analysis of the variables.

RESULTS

Out of 86 patients, 6 patients were lost follow-up and 5 patients developed non procedural related mortality in follow-up. All baseline characteristics of study population were shown in Table 1. The BP, antihypertensive medication, serum creatinine and GFR data compared at preprocedure and follow-up period in Table 2. There was no procedure related mortality. Two patients had local vascular complications which were managed conservatively. Out of 86 patients, 83 patients had atherosclerosis RAS and 3 patients takayasu arteritis.

The mean systolic BP was reduced from 170.15 ± 20.10 to 146.60 ± 17.32 mmHg and diastolic BP from 98.38 ± 10.55 to 89.88 ± 9.22 mmHg at one mo follow-up. This significant reduction in BP after PTRA was maintained at 6 mo follow up of 144.23 ± 18.19 systolic and 88.26 ± 9.79 diastolic BP respectively (Table 1). There was a statistically significant reduction in systolic BP compared to pre-intervention (paired *t* test: *P* <

Group[1]	*n*	mean ± SD	*P* value
Age (yr)			0.51
A	26	56.81 ± 13.87	
B	49	54.80 ± 11.55	
Initial systolic BP (mmHg)			0.01
A	26	179.31 ± 20.32	
B	49	166.00 ± 18.76	
Initial diastolic BP (mmHg)			0.04
A	26	101.92 ± 10.90	
B	49	96.79 ± 10.14	
Initial mean BP (mmHg)			0.01
A	26	141.00 ± 17.73	
B	49	129.00 ± 16.76	
No. of medications			0.01
A	26	3.26 ± 0.77	
B	49	2.87 ± 0.57	
Creatinine (mg/dL)			0.07
A	26	1.38 ± 0.48	
B	49	1.11 ± 0.66	
Diameter of stent			0.23
A	26	5.76 ± 0.94	
B	49	6.05 ± 0.98	
Percent of renal artery stenosis (RAS)			0.05
A	26	87.65 ± 7.71	
B	49	83.79 ± 8.40	
GFR (mL/min)			0.01
A	26	54.03 ± 24.22	
B	49	72.97 ± 25.43	
Duration of HT (yr)			0.55
A	26	4.00 ± 3.96	
B	49	3.40 ± 3.32	

Values are presented as mean ± SD. [1]Group A: Patients who did not show blood pressure reduction after percutaneous transluminal renal artery (PTRA) (26 patients); group B: Patients who showed blood pressure reduction after PTRA (49 patients). HT: Hormone Therapy; BP: Blood pressure; GFR: Glomerular filtration rate.

0.0001). The 65.33% of patients showed reduction in BP. There was no difference in the magnitude of systolic BP response among patients treated for bilateral RAS compared with those treated for unilateral RAS.

Mean intake of total number of medicines at baseline was 3.07 ± 0.69. At 1 mo follow-up, number of medicines reduced to 2.37 ± 0.84 and at 6 mo to 2.25 ± 0.94. There was statistically significant reduction in mean number of medicines intake ($P < 0.0001$).

At baseline, mean serum creatinine value was 1.21 ± 0.66 mg/dL. After PTRA and stenting, at 48 h there was mild elevation in serum creatinine to 1.29 ± 0.88 mg/dL. At 1 mo of follow up, serum creatinine was 1.33 ± 1.27 mg/dL and at 6 mo was 1.21 ± 0.79 mg/dL. There was no statistically significant difference in serum creatinine value after PTRA. At baseline mean GFR was 65.71 ± 25.20 mL/min. After PTRA and stenting at 6 mo of follow up, GFR was 66.68 ± 25.03 mL/min. There was no statistically significant difference in GFR at follow up after PTRA and stenting (Table 1).

PTRA and stenting to renal artery significantly lowers BP and mean number of drug intake but not cause signif-

		Group A *n* = 26	Group B *n* = 49	*P* value
	Male gender	24 (92.3)	33 (67.3)	0.016
	Smoker	22 (84.6)	33 (67.4)	0.1
	Ischemic heart disease	21 (80.8)	35 (71.4)	0.376
	Diabetes mellitus	8 (30.8)	19 (38.8)	0.49
	C/f of LVF	5 (19.2)	11 (22.4)	0.746
	Smoking	22 (84.6)	33 (67.4)	0.1
	Renal bruit	20 (76.9)	29 (59.2)	0.124
	Refractory HT	23 (88.5)	37 (75.5)	0.182
	LVH	14 (53.9)	14 (28.6)	0.031
	LAD	19 (73.1)	34 (69.4)	0.733
	TVD	12 (46.2)	13 (26.5)	0.08
Renal artery stenosis (unilat *vs* bilat)	Unilateral RAS	15 (57.7)	40 (81.6)	0.026
	Bilateral RAS	11 (42.3)	9 (18.4)	
LMCA disease	Absent	23 (88.5)	47 (95.9)	0.218
	Present	3 (11.5)	2 (4.1)	

Group A: Patients who did not show blood pressure reduction after percutaneous transluminal renal artery (PTRA) (26 patients); and group B: Patients who showed blood pressure reduction after PTRA (49 patients). HT: Hormone therapy; LVH: Left ventricular hypertrophy; TVD: Triple vessel disease; LAD: Left anterior descending; LVF: Left ventricular function; RAS: Renal artery stenosis; LMCA: Left main coronary artery.

icantly reduction in serum creatinine or change in GFR.

Prediction of BP reduction after PTRA among resistant hypertensive patients

In order to evaluate predictors of poor BP reduction after successful PTRA and stenting, we divided 75 patients into two groups: group A, the non-responsive group, which included patients without significant BP reduction after PTRA (26 patients), and group B, the responsive group, which included patients who showed significant BP reduction followed PTRA (49 patients) (BP reduction < 140/90 mmHg with or without drugs was considered significant reduction).

Higher baseline systolic and diastolic BP (number value < 0.01 and < 0.04, respectively) and higher mean intake of no. of medications (P value < 0.01) for control of BP was associated with poor response of BP control after successful PTRA and stenting. Non-responsive group associated with higher mean baseline serum creatinine (1.38 mg/dL *vs* 1.11 mg/dL) but not statistically significant ($P = 0.07$). But baseline low GFR < 60 mL/min was associated with poor response after PTRA and stenting ($P < 0.01$). Higher initial % of RAS was also associated with poor response ($P = 0.05$). Between these groups, neither duration of Hormone Therapy nor diameter of stent used was significantly different (Table 3).

Clinical features in the resistant hypertensive group

Comparing various characteristics between both groups reveals male sex ($P = 0.016$), left ventricular hypertrophy ($P = 0.031$), presence of triple vessel disease ($P = 0.08$)

Table 5 Multivariate analysis: The independent predictors for poor blood pressure response after percutaneous transluminal renal artery				
Variables	**Univariate *P* value**	**Multivariate analysis**		**95%CI**
		***P* value**	**β**	**Exp β**	
Male sex	0.02	0.046	1.797	6.032	1.028–35.380
High mean SBP	0.01	NS			
High mean DBP	0.05	NS			
High mean BP	0.09	0.013	-0.044	0.957	0.925–0.991
Low GFR	0.01	0.015	1.377	3.965	1.308–12.020
(< 60 mL/min)					
LVH	0.03	NS			
Drugs (*n*)	0.01	NS			
Bilateral *vs*	0.02	NS			
unilateral RAS					
Percent of stenosis	0.06	NS			
Presence of TVD	0.09	NS			
Constant			2.365	10.65	

TVD: Triple vessel disease; BP: Blood pressure; RAS: Renal artery stenosis; GFR: Glomerular filtration rate; LVH: Left ventricular hypertrophy; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; NS: Not significant.

and presence of bilateral RAS ($P = 0.026$) were associated with poor outcome after PTRA and stenting (Table 4).

DISCUSSION

In the current study we demonstrated that in patients with significant RAS, PTRA improved BP control and decreased mean number of drug intake significantly and this improvement was maintained during the entire follow-up period of 6 mo. PTRA and stenting did not cause significant improvement in renal function (*P* value for both serum creatine and GFR was not significant).

In recent years, role of PTRA and stenting in management of RAS has been questioned. In the early 1980s the concept was that revascularization of the stenotic atherosclerotic renal artery will salvage the ischemic kidney and will cure hypertension[23]. Revascularization methods and medication have improved considerably over the past 20 years and the aims of managing patients with atherosclerotic renal artery stenosis (ARAS) have progressed from focusing on BP control to stabilizing renal function and finally to preventing clinical events. However, as the procedure became broadly applied during the 1990s mixed results emerged. Some patients showed major benefit after PTRA, while others experienced further deterioration of renal function and major morbidity[24]. Today it is acknowledged that ARAS is a complex clinical entity that ranges from asymptomatic disease discovered incidentally on imaging to high grade bilateral disease complicated by recurrent pulmonary edema, severe hypertension, and progressive renal failure. RAS is generally associated with high incidence of associated CAD and target organ damage. Mortality in these patients is high and mostly related to cardiovascular events regardless of whether renal revascularization was performed[25].

In recent years several controlled trials were designed to evaluate the effectiveness of PTRA *vs* medical treatment in patients with severe ARAS. The DRASTIC study included a cohort of 106 hypertensive subjects with ARAS. The patients were randomly assigned to revascularization or medical treatment, but after 12 mo of follow-up no difference in BP control or renal function was demonstrated between the groups[26]. The STAR study, which included 140 patients with creatinine clearance < 80 mL/min per 1.73 m^2 and ARAS $\geq$ 50%, also failed to show benefit of the invasive approach *vs* medical treatment[27].

The largest randomized trial, the ASTRAL study, comparing revascularization to medical treatment for ARAS, examined 806 subjects who were followed for 5 years. This study concluded that revascularization for ARAS has more risk than benefit[23]. But important limitation of the trial was the selected population. Patients were enrolled in the trial only if their own physician was uncertain as to whether revascularization would provide a worthwhile clinical benefit. Patients with symptomatic ARAS such as uncontrolled hypertension despite optimal medical treatment, or with recurrent episodes of flush pulmonary edema were not included in the study[23]. This study is at the top of the list with ASTRAL, raised considerable debate regarding the management of patients with ARAS[28]. The main claim of the ASTRAL critics was that the success of PTRA for ARAS is strongly dependent on the selection of the right patients for this procedure.

In our study, we found PTRA and stenting associated with significant improvement in BP control with reduced mean intake of drugs without improvement in renal function. We also sought predictors of poor BP control after successful PTRA and stenting.

The predictors of poor response to BP control after PTRA and stenting by univariate analysis were male sex, high baseline systolic, diastolic and mean BP, low GFR, presence of LVH, high baseline mean intake of number of drugs, presence of bilateral stenosis, higher angiographic % diameter of stenosis and presence of TVD. But on multivariate analysis; the independent predictors for poor BP response after PTRA were male sex ($P = 0.046$), higher baseline mean BP ($P = 0.013$) and low GFR (< 60 mL/min) ($P = 0.015$) (Table 5).

Patients with poor BP response (34.66%) may be considered for renal sympathetic denervation therapy. As early studies in animals and in humans suggested that the renal nerves play a role in BP regulation. A series of pilot studies as well as a clinical trial (symplicity HTN-2) involving patients with uncontrolled hypertension then showed that a catheter-based system can safely denervate the kidney and produce notable and sustained reductions in BP. Ongoing symplicity HTN-3: Renal Denervation in Patients With Uncontrolled Hypertension trial will help us to establish whether therapeutic renal denervation using a catheter-based approach is a safe and effective therapy for patients with uncontrolled hypertension.

Study limitations

Given that the majority of patients were Asian, the find-

ings in our trial may not be generalized among other ethnic and racial populations. Another limitation of this study is that we have not used any emboli protection device. As atheroembolism is major concern in percutaneous intervention of renal artery and associated with different degree of renal impairment. Atheroembolism may impair outcome of PTRA and stenting particularly on renal function. Use of distal embolic protection device may be associated with improved outcome. We have not used FFR to evaluate lesion severity in our study. FFR can predict individual response to renal artery stenting and improve outcome of PTRA and stenting.

In conclusion, considering the results of our study and previous works it appears that the main effect of renal artery revascularization in ARAS is on BP control in patients with resistant hypertension, with minimal influence on renal function. Male sex, higher baseline BP and low GFR (< 60 mL/min) are associated with poor BP response after successful PTRA and stenting. Further studies with emboli protection devices and FFR to assess severity of lesion may be helpful to validate this observation.

COMMENTS

Background
Percutaneous transluminal renal artery angioplasty (PTRA) and stenting is an established procedure for the treatment of renovascular hypertension caused by renal artery stenosis (RAS). Recently published trials have questioned the efficacy of PTRA and stenting of renal artery.

Research frontiers
The PTRA technique has become the cornerstone for treatment of RAS and is now the first line treatment to be proposed. Balloon angioplasty alone was first proposed but several series reported the successful use of endovascular stents for treating suboptimal angioplasty results and as a primary intervention for atherosclerotic lesions and particularly ostial lesions with better immediate and long-term results than with balloon angioplasty alone.

Innovations and breakthroughs
They found PTRA and stenting associated with significant improvement in blood pressure (BP) control with reduced mean intake of drugs without improvement in renal function. Authors also sought predictors of poor BP control after successful PTRA and stenting.

Peer review
This is a study on percutaneous transluminal renal artery dilatation and stenting in a cohort of 86 patients with significant renal artery stenosis.

REFERENCES

1   **Wolak T**, Belkin A, Ginsburg V, Greenberg G, Mayzler O, Bolotin A, Paran E, Szendro G. Does percutaneous transluminal renal artery angioplasty improve blood pressure control and renal function in patients with atherosclerotic renal artery stenosis? *Isr Med Assoc J* 2011; **13**: 619-624 [PMID: 22097232]

2   **Jaff MR**, Bates M, Sullivan T, Popma J, Gao X, Zaugg M, Verta P. Significant reduction in systolic blood pressure following renal artery stenting in patients with uncontrolled hypertension: results from the HERCULES trial. *Catheter Cardiovasc Interv* 2012; **80**: 343-350 [PMID: 22511402 DOI: 10.1002/ccd.24449]

3   **Rihal CS**, Textor SC, Breen JF, McKusick MA, Grill DE, Hallett JW, Holmes DR. Incidental renal artery stenosis among a prospective cohort of hypertensive patients undergoing coronary angiography. *Mayo Clin Proc* 2002; **77**: 309-316 [PMID: 11936924 DOI: 10.4065/77.4.309]

4   **Henry M,** Henry I, Klonaris C, Polydorou A, Rath P, Lakshmi G, Rajacopal S, Hugel M. Renal angioplasty and stenting under protection: the way for the future? *Journal of the Institute of Cardio Vascular Diseases Disease* 2007; **1**: 1-12

5   **Missouris CG**, Buckenham T, Cappuccio FP, MacGregor GA. Renal artery stenosis: a common and important problem in patients with peripheral vascular disease. *Am J Med* 1994; **96**: 10-14 [PMID: 8304356 DOI: 10.1016/0002-9343(94)90109-0]

6   **Gross CM**, Krämer J, Waigand J, Uhlich F, Olthoff H, Luft FC, Dietz R. Ostial renal artery stent placement for atherosclerotic renal artery stenosis in patients with coronary artery disease. *Cathet Cardiovasc Diagn* 1998; **45**: 1-8 [PMID: 9736342 DOI: 10.1002/(SICI)1097-0304(199809)45]

7   **Jean WJ**, al-Bitar I, Zwicke DL, Port SC, Schmidt DH, Bajwa TK. High incidence of renal artery stenosis in patients with coronary artery disease. *Cathet Cardiovasc Diagn* 1994; **32**: 8-10 [PMID: 8039226 DOI: 10.1002/ccd.1810320103]

8   **Schreiber MJ**, Pohl MA, Novick AC. The natural history of atherosclerotic and fibrous renal artery disease. *Urol Clin North Am* 1984; **11**: 383-392 [PMID: 6464247 DOI: 10.1007/BF01576887]

9   **Strandness DE**. Natural history of renal artery stenosis. *Am J Kidney Dis* 1994; **24**: 630-635 [PMID: 7942821]

10 **Zierler RE**. Screening for renal artery stenosis: is it justified? *Mayo Clin Proc* 2002; **77**: 307-308 [PMID: 11936923 DOI: 10.4065/77.4.307]

11 **Safian RD**, Textor SC. Renal-artery stenosis. *N Engl J Med* 2001; **344**: 431-442 [PMID: 11172181 DOI: 10.1056/NEJM200102083440607]

12 **Olin JW**. Atherosclerotic renal artery disease. *Cardiol Clin* 2002; **20**: 547-562, vi [PMID: 12472042 DOI: 10.1016/S0733-8651(02)00091-7]

13 **Novick AC**, Ziegelbaum M, Vidt DG, Gifford RW, Pohl MA, Goormastic M. Trends in surgical revascularization for renal artery disease. Ten years' experience. *JAMA* 1987; **257**: 498-501 [PMID: 3795433 DOI: 10.1001/jama.1987.03390040114028]

14 **Weibull H**, Bergqvist D, Bergentz SE, Jonsson K, Hulthén L, Manhem P. Percutaneous transluminal renal angioplasty versus surgical reconstruction of atherosclerotic renal artery stenosis: a prospective randomized study. *J Vasc Surg* 1993; **18**: 841-850; discussion 850-852 [PMID: 8230572 DOI: 10.1016/0741-5214(93)90340-R]

15 **Cambria RP**. Surgery: Indications and Variables that Affect Procedural Outcome, as well as Morbidity and Mortality. *J Invasive Cardiol* 1998; **10**: 55-58 [PMID: 10762767]

16 **van de Ven PJ**, Kaatee R, Beutler JJ, Beek FJ, Woittiez AJ, Buskens E, Koomans HA, Mali WP. Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: a randomised trial. *Lancet* 1999; **353**: 282-286 [PMID: 9929021 DOI: 10.1016/S0140-6736(98)04432-8]

17 **Henry M**, Amor M, Henry I, Ethevenot G, Tzvetanov K, Courvoisier A, Mentre B, Chati Z. Stents in the treatment of renal artery stenosis: long-term follow-up. *J Endovasc Surg* 1999; **6**: 42-51 [PMID: 10088889 DOI: 10.1583/1074-6218(1999)006<0042]

18 **Henry M**, Amor M, Henry I, Ethevenot G, Allaoui M, Tricoche O, Porte JM, Touchot N. Stent placement in the renal artery: three-year experience with the Palmaz stent. *J Vasc Interv Radiol* 1996; **7**: 343-350 [PMID: 8761809 DOI: 10.1016/S1051-0443(96)72864-6]

19 **Blum U**, Krumme B, Flügel P, Gabelmann A, Lehnert T, Buitrago-Tellez C, Schollmeyer P, Langer M. Treatment of ostial renal-artery stenoses with vascular endoprostheses after unsuccessful balloon angioplasty. *N Engl J Med* 1997; **336**: 459-465 [PMID: 9017938 DOI: 10.1056/NEJM199702133360702]

20 **van de Ven PJ**, Beutler JJ, Kaatee R, Beek FJ, Mali WP, Geyskes GG, Koomans HA. Transluminal vascular stent for ostial atherosclerotic renal artery stenosis. *Lancet* 1995; **346**: 672-674

[PMID: 7658821 DOI: 10.1016/S0140-6736(95)92283-0]

21   **Dorros G**, Jaff M, Jain A, Dufek C, Mathiak L. Follow-up of primary Palmaz-Schatz stent placement for atherosclerotic renal artery stenosis. *Am J Cardiol* 1995; **75**: 1051-1055 [PMID: 7747688 DOI: 10.1016/S0002-9149(99)80723-1]

22   **Wheatley K**, Ives N, Gray R, Kalra PA, Moss JG, Baigent C, Carr S, Chalmers N, Eadington D, Hamilton G, Lipkin G, Nicholson A, Scoble J. Revascularization versus medical therapy for renal-artery stenosis. *N Engl J Med* 2009; **361**: 1953-1962 [PMID: 19907042 DOI: 10.1056/NEJMoa0905368]

23   **Novick AC**, Pohl MA, Schreiber M, Gifford RW, Vidt DG. Revascularization for preservation of renal function in patients with atherosclerotic renovascular disease. *J Urol* 1983; **129**: 907-912 [PMID: 6854759]

24   **Textor SC**, Wilcox CS. Renal artery stenosis: a common, treatable cause of renal failure? *Annu Rev Med* 2001; **52**: 421-442 [PMID: 11160787 DOI: 10.1146/annurev.med.52.1.421]

25   **Conlon PJ**, Little MA, Pieper K, Mark DB. Severity of renal vascular disease predicts mortality in patients undergoing coronary angiography. *Kidney Int* 2001; **60**: 1490-1497 [PMID:

11576364 DOI: 10.1046/j.1523-1755.2001.00953.x]

26   **van Jaarsveld BC**, Krijnen P, Pieterman H, Derkx FH, Deinum J, Postma CT, Dees A, Woittiez AJ, Bartelink AK, Man in 't Veld AJ, Schalekamp MA. The effect of balloon angioplasty on hypertension in atherosclerotic renal-artery stenosis. Dutch Renal Artery Stenosis Intervention Cooperative Study Group. *N Engl J Med* 2000; **342**: 1007-1014 [PMID: 10749962 DOI: 10.1056/NEJM200004063421403]

27   **Bax L**, Woittiez AJ, Kouwenberg HJ, Mali WP, Buskens E, Beek FJ, Braam B, Huysmans FT, Schultze Kool LJ, Rutten MJ, Doorenbos CJ, Aarts JC, Rabelink TJ, Plouin PF, Raynaud A, van Montfrans GA, Reekers JA, van den Meiracker AH, Pattynama PM, van de Ven PJ, Vroegindeweij D, Kroon AA, de Haan MW, Postma CT, Beutler JJ. Stent placement in patients with atherosclerotic renal artery stenosis and impaired renal function: a randomized trial. *Ann Intern Med* 2009; **150**: 840-848, 840-848 [PMID: 19414832]

28   **White CJ.** Kiss my astral: one seriously flawed study of renal stenting after another. *Catheter Cardiovasc Interv* 2010; **75**: 305-307 [PMID: 20095015 DOI: 10.1002/ccd.22416]