Does Percutaneous Transluminal Renal Artery Angioplasty Improve Blood Pressure Control and Renal Function in Patients with Atherosclerotic Renal Artery Stenosis?

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ABSTRACT: **Background:** Percutaneous angioplasty (PTA) and stenting is an established procedure for the treatment of hypertension caused by atherosclerotic renal artery stenosis. However recently, the decision whether or not to perform this procedure has raised considerable debate.

Objectives: To examine the association between the basic clinical and radiological characteristics of candidates for renal artery PTA and the clinical outcome of the procedure in terms of improvement of blood pressure control and renal function.

Methods: We conducted a retrospective cohort study of all patients who underwent percutaneous transluminal renal artery angioplasty (PTRA) and stent implantation in a tertiary medical center during the period 2000–2007. The clinical and radiological data were extracted from the medical file of each patient. Blood pressure measurements and creatinine level were recorded before the procedure and 1 month, 6 months, 12 months and 18 months after PTRA.

Results: Thirty-two patients were included in the final statistical analysis. The mean age of the study population was 66.6 ± 8.8 years old and 75% were men. There was a significant reduction in both systolic and diastolic blood pressure 1 month after the procedure: 160.5 ± 24.7 vs. 141.8 ± 23.6 mmHg and 83.8 ± 12.9 vs. 68.8 ± 11.8 mmHg respectively ($P < 0.001$). The reduction in blood pressure was constant throughout the follow-up period and was evident 18 months after the procedure: 160.5 ± 24.7 vs. 135.0 ± 35.1 mmHg and 83.8 ± 12.9 vs. 71.3 ± 16.5 mmHg respectively ($P < 0.001$). However, no improvement in renal function was observed at any time during the follow-up period. We could not demonstrate an association between clinical or radiological features and the clinical outcome after PTRA.

Conclusions: Our findings show that PTRA can be considered an effective procedure for improving blood pressure control in patients with atherosclerotic renal artery stenosis (ARAS) and resistant hypertension. This research, together with previous studies, strengthens the knowledge that the decline in glomerular filtration rate seen in many patients with ARAS is non-reversible and is not improved by PTRA.

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The last decade has seen substantial improvement in non-invasive radiological diagnosis of atherosclerotic disease, including atherosclerotic renal artery disease. However, the improvement in diagnosis occurred in atherosclerotic renal artery stenosis but not in renovascular hypertension. Currently, the diagnosis of renovascular hypertension is made retrospectively [1]. Nevertheless, the increase in the number of diagnoses of renal artery stenosis led to an increase in procedures aiming to improve renal perfusion. The assumption was that restoring renal blood flow will improve both blood pressure control and renal function [2]. Unfortunately, this hypothesis did not prove to be entirely correct [3,4]. Various prospective trials raised the question whether percutaneous transluminal renal artery angioplasty should be performed and which patients would benefit most [5,6]. To answer these questions several measurements were evaluated as predictors of the efficacy of PTRA: pre-procedure renal function, plasma renin activity, the degree of renal artery stenosis, and renal resistance index [7]. Renal resistance index, measured by duplex ultrasound, is an indirect parameter that is determined by obtaining Doppler wave curves from the intrarenal segmental arteries. RI is calculated using the formula: RI = peak systolic flow velocity-diastolic flow velocity/peak systolic flow velocity [8].

The result of renal artery stenosis with normal renal parenchyma is decreased RI compared to RI of a kidney with a non-stenotic artery. However, any intrinsic renal parenchymal disease will increase the RI level. Several studies demonstrated that RI above 0.8 is associated with failure of PTRA to improve renal function and blood pressure control [9], but other trials failed to show this relationship [10].

The aim of our study was to examine the clinical and radiological features of candidates for PTRA and the clinical outcome of the procedure, focusing on the changes in renal function and blood pressure control.

PTRA = percutaneous transluminal renal artery angioplasty
RI = resistance index

PATIENTS AND METHODS

This retrospective cohort study related to all patients who underwent percutaneous transluminal renal artery angioplasty and stent implantation in a tertiary medical center during the period 2000–2007. Inclusion criteria for participation in the study were: a) diagnosis of ARAS using duplex sonography, b) PTRA with stent insertion, and c) 18 months follow-up after the procedure. The exclusion criteria were: a) non-atherosclerotic renal artery stenosis, b) unsuccessful PTRA, and c) lost to follow-up.

DATA COLLECTION

The clinical and demographic data were obtained from the patient's medical files. The following data were extracted: age, gender, presence of atherosclerotic risk factors, and atherosclerotic organ damage; measurements of blood pressure and plasma creatinine before the procedure, post-PTRA and during the follow-up period; and antihypertensive treatment before and after PTRA. The results of the sonographic study included percentage of stenosis, resistance index, acceleration time, aortic velocity, renal artery velocity and renal-aortic ratio. Data from other imaging studies (computed tomography angiography and magnetic resonance angiography) were also included.

The study group was divided into three groups according to the indication for PTRA with stent insertion:

- Group 1 – patients with resistant hypertension. Resistant hypertension was defined as failure to reach target blood pressure with treatment of at least three antihypertensive medications in adequate doses (the medical regimen should include diuretics) [11]
- Group 2 – patients with deterioration of renal function
- Group 3 – patients who had both resistant hypertension and impaired renal function.

STATISTICAL ANALYSIS

Statistical analysis was performed using the SPSS software. Since the nature of the variables involved in our study was either continuous or categorical, we used one-sample (or two independent samples) *t*-test, one-way repeated measures ANOVA test, and chi-square test (or Fisher's exact test). In cases where we did not assume that the dependent variable was a normally distributed interval variable, we used the Wilcoxon-Mann-Whitney test or the Kruskal-Wallis test (if there were more than two independent variables). In accordance with common practice we considered $P = 0.05$ statistically significant. The study was approved by the ethics committee of our institute.

ARAS = atherosclerotic renal artery stenosis

RESULTS

For the period 2000–2007 we identified 41 patients as candidates for PTRA, of whom 9 were excluded from the study. In two of the nine patients PTRA was performed because of a diagnosis other than ARAS: Takayasu arteritis in one and renal cell carcinoma in the other; two patients did not undergo PTRA due to their poor medical condition and five patients were lost to follow-up. Thus, 32 patients met the inclusion criteria of the study and completed 18 months follow-up.

CLINICAL CHARACTERISTICS [TABLE 1]

The mean age of the study population was 66.6 ± 8.8 years and 75% were males. The indication for performing PTRA [11,12] in the study population was resistant hypertension to medical treatment in 12 patients (37.5%), progressive dete-

Table 1. Baseline characteristics of study population: clinical, laboratory and imaging data

	All patients (N=32)
Male gender	24 (75%)
Age (mean, yrs)	66.6 ± 8.8
Body mass index (mean, kg/m²)	27.6 ± 3.1
Smoking	12 (38%)
Background diseases	
Chronic renal failure	19 (59.3%)
Peripheral artery disease	15 (46.8%)
Diabetes mellitus	14 (43.7%)
Chronic ischemic heart disease	17 (53.1%)
Congestive heart failure	5 (15.6%)
Cerebrovascular disease	15 (46.8%)
Aortic disease	8 (25.0%)
Dyslipidemia	27 (84.4%)
Flush pulmonary edema	2 (6.2%)
Blood pressure (mmHg)	
Systolic (mean)	160.5 ± 24.7
Diastolic (mean)	83.6 ± 13.5
Antihypertensive drugs (mean no. of drugs)	3.2 ± 0.9
Indication criteria	
Hypertension resistant to standard medication	12 (37.5%)
Renal function impairment	11 (34.3%)
Both of the above	9 (28.1%)
On antiplatelet treatment	30 (93.7%)
On statin treatment	24 (75.0)
Serum creatinine (mean, mg/dl)	1.52 ± 0.7
Serum hemoglobin (mean, mg/dl)	13.0 ± 1.7
Kidney size on ultrasound (mean, cm)	9.7 ± 0.9
Multiple renal arteries	5 (12.5%)
Stenosis	
Bilateral	17 (53.1%)
› 60%	81.8%
‹ 60%	48.8%
Doppler sonography	
Kidney size (mean, cm)	10.4 ± 1.3
Resistance index (=1-edv/psv)	0.66 ± 0.1
Acceleration time (mean, msec)	88.1 ± 33
Aortic velocity (mean, cm/sec)	84.5 ± 19.7
Renal-aortic ratio (mean)	5.4 ± 2.6

edv/psv = peak systolic velocity/end diastolic velocity

rioration of renal function in 11 patients (34.3%) (group 2), and both of the above in 9 patients (28.1%) [9]. The mean systolic and diastolic blood pressure before the procedure was 160.5 ± 24.7 and 83.6 ± 13.5 mmHg respectively.

LABORATORY AND SONOGRAPHIC DATA [TABLE 1]

The mean serum creatinine level was 1.52 ± 0.7 mg/dl. Bilateral renal artery stenosis was detected in 17 patients (53.1%). In 81.8% of the patients a stenosis of more than 60% in the renal artery was observed. The mean RI of the kidney supplied by the stenotic artery was 0.6 ± 0.1. The mean acceleration time was 88.1 ± 33.0 msec, and the mean renal aortic ratio was 5.4 ± 2.6.

PROCEDURE

Thirty-two patients underwent PTRA. The procedure was performed on 48 arteries: on 1 artery in 17 patients, on 2 arteries in 14 patients, and on 3 arteries in 1 patient. ULTRAVIST contrast media was used, with a mean volume of 71.2 ml. In all patients predilatation of the artery was performed followed by the stent insertion. Post-PTRA stenosis was detected in six patients 6 months after the procedure; four patients had restenosis of less than 60% and two had more than 60%. In these two patients PTRA was repeated. Complications included bleeding in two patients (6.2%): retroperitoneal hematoma in one and inguinal bleeding in the other. No major complications or death occurred in the first month following the procedure.

BP: PRE-PROCEDURE COMPARED TO FOLLOW-UP PERIOD [TABLE 2]

Blood pressure was measured at the following time intervals: pre-procedure, and 1, 6, 12, and 18 months after the procedure. There was a significant decrease in blood pressure levels 1 month after the PTRA: 160.5 ± 24.7/83.6 ± 13.5 mmHg versus 141.8 ± 23.5/68.8±11.8 mmHg respectively ($P < 0.001$). The decrease in blood pressure was sustained throughout the 18 months of the follow-up period. The last blood pressure measurement (18 months post-PTRA) remained significantly lower compared to pre-procedure values (160.5 ± 24.7/83.6 ± 13.5 mmHg vs. 135.0 ± 35.0/71.3 ± 16.5 mmHg respectively, $P < 0.001$). Although the blood pressure decreased further during the follow-up period, this additional reduction compared to the decline in the first month after PTRA was not significant.

In contrast to blood pressure, PTRA did not change significantly, nor did the number of antihypertensive medications or the levels of serum creatinine.

PREDICTION OF BP REDUCTION AFTER PTRA AMONG RESISTANT HYPERTENSIVE PATIENTS

In order to evaluate which of the pre-procedure characteristics might predict the efficiency of PTRA in blood pressure control, we divided the 21 patients whose indication for PTRA was resistant hypertension into two groups: group A – the non-responsive group, which included patients with no blood pressure reduction after PTRA (9 patients), and group B – the responsive group, which included patients who showed significant blood pressure reduction following PTRA (12 patients).

BP = blood pressure

Table 2. Blood pressure, antihypertensive medication and serum creatinine initial versus follow-up measurements

					95% confidence interval for mean		
		Time of follow-up	Mean	SD	Lower bound	Upper bound	*P* value
Systolic blood pressure	0	Pre-procedure	160.5	24.7	155.94	172.01	
	1	1 mo	141.79	23.565	132.65	150.92	
	2	6 mos	140.28	26.786	130.09	150.46	0.00
	3	12 mos	135.37	31.051	120.40	150.33	
	4	18 mos	135.00	35.063	114.76	155.24	
Diastolic blood pressure	0	Pre-procedure	83.6	13.5	79.65	88.04	
	1	1 mo	68.82	11.842	64.23	73.41	
	2	6 mos	69.21	13.478	64.08	74.33	0.00
	3	12 mos	67.00	15.979	59.30	74.70	
	4	18 mos	71.29	16.518	61.75	80.82	
No. of antihypertensive drugs	0	Pre-procedure	3.20	.966	2.89	3.51	
	1	1 mo	2.95	1.161	2.42	3.48	
	2	6 mos	2.80	1.152	2.26	3.34	0.74
	3	12 mos	3.00	1.348	2.14	3.86	
	4	18 mos	2.91	1.300	2.04	3.78	
Serum creatinine (mg/dl)	0	Pre-procedure	1.643	0.7783	1.388	1.899	
	1	1 mo	1.588	0.6902	1.233	1.943	
	2	6 mos	1.633	0.6572	1.269	1.997	0.99
	3	12 mos	1.694	0.7366	1.128	2.261	
	4	18 mos	1.672	0.9545	1.031	2.313	

Values are presented as mean ± SD

● **BP AND INITIAL RENAL CHARACTERISTICS** [TABLE 3]

Neither initial blood pressure nor renal function was significantly different between these two groups. However, baseline creatinine level showed a tendency to increase in the non-responsive group versus the responsive group (1.54 ± 0.59 vs. 1.18 ± 0.29 mg/dl, respectively, $P = 0.1$).

● **CLINICAL FEATURES IN THE RESISTANT HYPERTENSIVE GROUP** [TABLE 4]

There was no significant clinical variation between the two groups. However, the incidence of hyperlipidemia was higher among patients in the non-responsive group: 9 (100%) compared to only 8 (66.7%) in the responsive group ($P = 0.054$).

Table 3. Blood pressure and initial renal characteristics of patients with resistant hypertension: responsive vs. unresponsive to PTRA

	Group*	N	Mean	SD	*P* value
Age (yrs)	A	9	64.44	11.226	0.37
	B	12	68.58	9.615	
BMI (kg/m2)	A	9	28.4711	6.16167	0.74
	B	12	27.8242	2.29888	
Initial systolic BP (mmHg)	A	9	172.78	20.783	0.91
	B	12	173.75	19.671	
Initial diastolic BP (mmHg)	A	9	88.33	8.660	0.44
	B	12	92.08	11.958	
No. of medications	A	9	3.22	.972	0.66
	B	12	3.00	1.206	
Creatinine (mg/dl)	A	9	1.1844	.29151	0.11
	B	12	1.5492	.59829	
Kidney size (cm)	A	4	10.888	.6356	0.33
	B	8	10.013	1.6296	
Resistance index	A	4	.668	.0670	0.48
	B	8	.639	.0629	
Acceleration time (sec)	A	4	81.00	30.299	0.91
	B	8	83.00	28.097	
Aortic velocity (cm/sec)	A	4	103.00	26.369	0.21
	B	8	88.25	13.360	
Renal-aortic ratio	A	4	3.978	2.0987	0.49
	B	6	4.608	.6037	

Values are presented as mean ± SD

*Group A – patients who did not show blood pressure reduction after PTRA (9 patients); group B – patients who showed blood pressure reduction after PTRA (12 patients)

Table 4. Clinical features of resistant hypertensive group: responsive vs. unresponsive to PTRA (rates)

		Group*				
		A (n=9)		B (n=12)		
		N	%	N	%	*P* value
	Male gender	6	66.7%	8	66.7%	0.99
	Smoker	4	44.4%	4	33.3%	0.26
	Ischemic heart disease	3	33.3%	4	33.3%	0.99
	Diabetes mellitus	2	22.2%	5	41.7%	0.35
	Congestive heart failure	1	11.1%	2	16.7%	0.72
	Peripheral vascular disease	3	33.3%	5	41.7%	0.23
	Cerbrovascular disease	5	55.6%	4	33.3%	0.31
	Hyperlipidemia#	9	100.0%	8	66.7%	0.054
	Renal failure	4	44.4%	6	50.0%	0.8
	Flush pulmonary edema	1	11.1%	0	.0%	0.237
	Bilateral renal artery stenosis	4	50.0%	4	33.3%	0.213
	Multiple renal arteries	3	33.3%	1	8.3%	0.16
	Aspirin treatment	9	100.0%	10	83.3%	0.19
	Statin treatment	7	77.8%	6	50.0%	0.19
Percentage of stenosis according to Doppler study	‹ 60%	2	50.0%	1	11.1%	0.12
	› 60%	2	50.0%	8	88.9%	
Type of stenosis seen on angiography	Osteal	4	57.1%	4	36.4%	0.38
	Non-osteal	3	42.9%	7	63.6%	
Resistant index	‹ 0.7	2	50.0%	5	62.5%	0.67
	≥ 0.7	2	50.0%	3	37.5%	

*Group A – patients who did not show BP reduction after PTRA (9 patients), and group B – patients who showed blood pressure reduction after PTRA (12 patients).

Triglycerides › 150 or low density lipoprotein › 130 mg/dl

BP CHANGES AFTER PTRA IN PATIENTS WITHOUT RESISTANT HYPERTENSION

Patients with the indication for PTRA of progressive deterioration of renal function and without resistant BP demonstrated a non-significant reduction in blood pressure during the first month following PTRA (135.9 ± 10.7/70.5 ± 6.9 vs. 126.6 ± 15.1/61.0 ± 70.1 mmHg, respectively). However, at the end of the study (18 months post-PTRA), blood pressure measurements returned to their initial value (143.2 ± 53.4/69.8 ± 22.7 mmHg).

DISCUSSION

In the current study we demonstrated that in patients with significant atherosclerotic renal artery stenosis and resistant hypertension, PTRA improved blood pressure control and this improvement was maintained during the entire follow-up period of 18 months. However, PTRA did not affect the number of antihypertensive medications that patients were taking, or renal function as reflected by serum creatinine levels. Patients with atherosclerotic renal stenosis but without resistant hypertension did not show improvement either in creatinine level or in blood pressure measurements.

The management of ARAS has been the focus of serious debate in recent years. In the early 1980s the concept was that revascularization of the stenotic atherosclerotic renal artery will salvage the ischemic kidney and will cure hypertension [13]. 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 [14]. 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. ARAS usually reflects the severity and

the extension of general atherosclerosis. It is also associated with atherosclerotic risk factors and target organ damage [15]. In view of the above, it is no surprise that patients with severe ARAS are usually older and have comorbid conditions [16]. Mortality in these patients is mostly related to cardiovascular events regardless of whether renal revascularization was performed [12].

In recent years several controlled trials were designed to examine the benefit of PTRA versus 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 months of follow-up no difference in blood pressure control or renal function was demonstrated between the groups [5]. The STAR study, which included 140 patients with creatinine clearance < 80 ml/min per 1.73 m^2 and ARAS ≥ 50%, also failed to show benefit of the invasive approach versus medical treatment [17]. 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 [6]. However, the authors emphasized that an 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 [6]. These studies, with ASTRAL at the top of the list, raised considerable debate regarding the management of patients with ARAS [18]. 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 observational study we sought to investigate what benefit PTRA would bring to the patients. Our major finding was that PTRA relieved the burden of blood pressure. Although there was no change in the use of several antihypertensive medications, blood pressure control was substantially improved. The other main finding was that PTRA failed to improve renal function. This observation of dissociation between blood pressure control and renal function following revascularization was previously demonstrated in renal transplant patients. The authors showed that revascularization of the stenotic renal artery of the transplant kidney reduced blood pressure by diminishing sodium and water retention. However, the correction of the stenotic artery brought no significant change to the glomerular filtration rate [19[.

Another study, published a year ago [20], reinforced both current and previous observations that relief of significant renal artery stenosis resulted in better control of blood pressure with no affect on renal function. Comparable to the current study the improvement in blood pressure control lasted for the entire study period.

ARAS does not fit the classical Goldblatt model for renovascular hypertension. Atherosclerosis is a disease that not only causes stenosis of the renal artery but probably affects the kidney and its small intra-renal arteries directly. It may be assumed that some patients with ARAS already suffer from irreversible renal damage and revascularization will not improve their renal function.

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 blood pressure control in patients with resistant hypertension, with minimal influence on renal function. Further studies are needed to validate this observation.

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