

Clinical efficacy of percutaneous renal revascularization with stent placement in hypertension among patients with atherosclerotic renovascular diseases

Seyed Mohammad Hassan Adel, Seyed Masood Syeidian, Mohammad Najafi,
Mohammad nourizadeh

*Department of Cardiology, Jondi Shapour Cardiovascular Research Centre, Imam Khomeini Hospital,
Jondishapour University of Medical Sciences, Ahwaz, Iran.*

Address for correspondence: *Dr. Seyed Mohammad Hassan Adel, Department of Cardiology,
Jondi Shapour Cardiovascular Research Centre, Imam Khomeini Hospital,
Azadegan Avenue, Jondishapour University of Medical Sciences, Ahwaz, Iran.
E-mail: dr.hassan.adel@gmail.com*

ABSTRACT

Aim: The aim was to assess the effect of renal angioplasty with stent on systolic, diastolic, and mean arterial blood pressure (MAP) in awake and sleep time with ambulatory blood pressure (ABP) monitoring (Holter monitoring). **Materials and Methods** Patients with angiographically proven atherosclerotic renal artery stenosis (RAS) were referred to the Angiography Department of Imam Hospital for intervention during a 1-year period from June 2008 to December 2009. Primary stent placement was attempted by a single operator in 27 severe RAS cases although 1 case was omitted from the study because of technical failure. Pre- and postprocedure creatinine levels, ejection fraction (EF), history of diabetes mellitus (DM), and ABP were obtained. Twenty-six (17 men, 9 women; average age, 62.6 years; age range, 90–21 years) consecutive patients participated in the study. **Results:** All patients had severe hypertension resistant to multiple medications; 10 patients had impaired renal function (serum creatinine level greater than 130 $\mu\text{mol/L}$). A total of 3 (11.5%) patients had congestive heart failure, and 10 (37.7%) were diabetic. Hypertension was cured in 1 (4%) patient, had improved in 23 (88.4%) patients, and had failed to respond to treatment in 2 (7.6%). Serum creatinine decreased significantly from 1.46 ± 0.89 to 1.35 ± 0.61 mg/dL ($P < 0.05$). **Conclusion:** Percutaneous transluminal angioplasty for atheromatous RAS rarely “cures” hypertension, but improved blood pressure control is often achieved.

Key words: Ambulatory blood pressure, atherosclerosis, hypertension

INTRODUCTION

Renal artery stenosis (RAS), mostly caused by atherosclerosis, can cause both renovascular hypertension and renal insufficiency.^[1,2] Treatment of RAS by surgery or balloon

angioplasty aims at avoiding lifelong antihypertensive treatment and progressive renal ischemia.^[1-3] The frequency of documented RAS varies from 0.5% to >20%, according to age^[4] and the thoroughness of investigation,^[5-7] and will probably increase with the increasing population age and the widespread use of noninvasive screening tests.^[1,3-8] Attempts at revascularization will also increase because angioplasty reported to be as effective as surgery^[9] allows treatment of older and more fragile patients. The efficacy and safety of angioplasty in hypertension among these patients should be objectively evaluated.^[9] With the exception of a randomized trial reported in an abstract form,^[10] only information based on retrospective analyses is

Access this article online

Quick Response Code:



Website:

www.jcdronline.com

DOI:

10.4103/0975-3583.78585

available.^[2,10,12,13] We compared the 1-month blood pressure (BP) outcome and the incidence of complications after diagnostic angiography plus angioplasty (angioplasty group) in patients with hypertension and unilateral atherosclerotic RAS. The number of antihypertensive agents required to obtain target BP was determined, and the BP outcome was documented with the use of 24-h ambulatory BP monitoring (ABPM), an observer-independent assessment that improves the repeatability of BP measurement.^[14]

MATERIALS AND METHODS

Patients were referred to the participating centers for hypertension and unilateral atherosclerotic RAS documented with intravenous subtraction angiography or a previous arteriography. Eligible patients were men and women younger than 90 years of age, with diastolic blood pressure (DBP) readings >90 mmHg on at least three occasions and/or receiving antihypertensive treatment; patients with a history of stroke, pulmonary edema, or myocardial infarction in the previous 6 months were not included. Anatomic inclusion criteria were determined from the qualifying angiography immediately before randomization. They comprised (1) the atherosclerotic nature of the RAS, as inferred from renal artery and aortic views; (2) a reduction in arterial diameter of either 70% without thrombosis; (3) a stenosis affecting the main renal artery, which had not been previously dilated; and (4) a functional kidney on the opposite side exhibiting a normal main artery or an arterial diameter reduction of <50%. Patients gave written informed consent before the qualifying angiography.

Clinical evaluation

The creatinine clearance was estimated from the serum creatinine (sCr) concentration with the Cockcroft formula^[15] and ABP was monitored over the 24 h before hospitalization. Patients were hospitalized for qualifying catheter angiography. Before randomization and at termination, 24-h ABP was measured with SpaceLabs 5300 or 90207 (SpaceLabs, Inc.) or Colin ABPM 630 (Colin Medical Instruments) monitoring devices programmed to record BP every 15 min during the day and every 30 min during the night. The same monitor was used on the same arm of each patient before randomization and at termination. Sitting BP was also measured with a mercury sphygmomanometer during each visit to compare DBP readings with oscillometric and ambulatory determinations.

Technique

The femoral approach was used in all cases less than 1 month

after selective angiography. The standard technique involved the placement of a 7-F introducer sheath into the femoral artery and negotiation of the stenosis by using a wire, and stents (EXPRESS (Boston Scientific Co), HERCULING (Abbot Vascular Co), and HIPPOCAMPUS (Invatec Co)) were positioned coaxially through a 7-F guiding catheter (RDC), with its tip in the renal ostium. Then 10–15 mL of the contrast material (Iopramide [150 mg of iodine per milliliter] was injected into the guiding catheter by using a side-arm adaptor to obtain fine adjustment of the stent position before deployment. In ostial lesions, stents were deliberately deployed so that they projected 1 mm into the aortic lumen. The optimal final stent diameter was determined by measuring the caliber of a normal segment of the same renal artery. Stenosis was not routinely predilated. The immediate technical result was evaluated with angiography. Technical success was defined as a residual stenosis of less than 10%. All patients received 5000 units of heparin that was administered intra-arterially during the procedure, and antiplatelet therapy (aspirin, 325 mg daily, and plavix, 450 mg loading and 75 mg daily for 2 months). No patients formally received anticoagulation therapy following this procedure. Intra-arterial angiographic follow-up was not performed routinely in this high-risk patient group. Clinical follow-up for 6–12 months was used to guide management. If patients showed clinical evidence of relapse following the initial favorable outcome, angiography was performed. Age, gender, pre- and postprocedure creatinine levels and ejection fraction, systolic and diastolic blood pressure recording, and history of DM were recorded. All patients were examined once within 3 weeks of the procedure. In most patients, blood pressure and sCr values were also available for as many as 3 months prior to the procedure. Renal impairment was classified as improved if the sCr level decreased by 10% or more and stable if it was within 10% of the preprocedure level. Renal impairment with an increase of more than 10% was classified as a failure. Hypertension was classified as cured if all antihypertension medication was stopped and diastolic blood pressure returned to less than 90 mmHg. Criteria for improvement were either diastolic blood pressure less than 90 mmHg without the increased medication dose or between 90 and 110 mmHg with a decrease greater than 15 mmHg and no increase in the medication dose. All other possibilities constituted a failure. Complications were evaluated by reviewing the renal angiography reports for immediate procedural complications and the case notes for delayed procedure-related and clinical complications.

Data analysis

We intended to enroll 27 patients. This sample size would have allowed 85% power to detect a 10 mmHg difference

in diastolic ABP at termination between the two groups, with a type I error of 5%. SPSS software was used for statistical analysis. Proportions were compared by the Proc Freq procedure and quantitative variables by the Proc T-test procedure.

RESULTS

Patients

Twenty-seven (18 men, 9 women; average age, 62.6 years; age range, 90–21 years) consecutive patients with angiographically proven atherosclerotic RAS were referred to the Angiography Department of Imam Hospital for renal arterial stenting treatment during a 1-year period from June 2008 to December 2009.

All patients had a history of severe hypertension with poor response to drug therapy; 10 (37.7%) patients had an impaired renal function (sCr level greater than 130 $\mu\text{mol/L}$ or 1.4 mg/dL), 3 (11.5%) had congestive heart failure, and 10 (37.7%) were diabetic. Primary stent placement was attempted by a single operator (there is one peripheral interventionalist in our center) in 27 severe RAS cases [Table 1].

Technical results

Technical success (primary patency) was achieved in 26 (96.2%) patients. Reasons for technical failure were failure to cross the stenosis with a guide wire in one patient.

Clinical outcome

One case was omitted from the study because of technical failure. Hypertension was cured in one (3.8%) patient, had

improved in 23 (88.4%) patients, and had failed to respond to treatment in 2 (7.8%) immediately after stenting clinically. Mean arterial systolic and diastolic blood pressures measured with Holter monitoring that recorded awake time, sleep time, and overall blood pressure for all patients who underwent stent placement for their hypertension (including no responders) are shown in Table 2. All blood pressure parameters (systolic/diastolic/mean, for overall, awake time, and sleep time) decreased significantly immediately after the intervention. After intervention in the female group, there was no significant difference for the decrease in mean/diastolic pressure especially in awake and sleep time ($P > 0.05$). In 46.1% of the patients, the mean arterial blood pressure control was improved after mean follow-up; it remained unchanged in 43% and deteriorated in 11% patients. Patients with diabetes also had improved blood pressure control [Table 3].

Renal impairment

Serum creatinine decreased significantly from 1.46 ± 0.89 to 1.35 ± 0.61 mg/dL ($P < 0.05$). Renal impairment improved in 12 (46.1%) patients ($>10\%$ decrease in the Cr concentration from baseline), stabilized in 12 (46.1%; 0–9% change in the Cr concentration), and failed to respond to treatment in 2 (7.8%; 10% increase in the Cr concentration). However, some important subgroup changes were noted.

Four (11%) patients had a deterioration of the renal function during 3 months after stenting, leading to chronic renal failure without leading to dialysis. Subgroup analysis in the improved renal function showed no significant change in sCr in patients with a normal renal function at baseline, but there was a significant ($P < 0.05$) decrease in sCr in patients with moderate and severe renal dysfunction, respectively [Table 4]. The more severe the renal dysfunction at baseline, the more the patients benefited from the

Table 1: Baseline clinical characteristics in patients before renal angioplasty

	Total [n = 26; n (%)]	Men [n = 17; n (%)]	Women [n = 9; n (%)]
Age (years)	62.6	64	60
Diabetes	10 (37.7)	7 (41)	3 (33)
Current smoker	3 (11)	3 (17)	0 (0)
Heart failure	3 (11.5)	1 (5.5)	2 (22.5)
Duration of hypertension > 5 year	20 (77)	15 (88)	5 (55)
Baseline creatinine > 1.3 mol/L	10 (37.7)	8 (47)	2 (22)
MSBP awake > 135 mmHg	26 (100)	17 (100)	9 (100)
MSBP sleep > 120 mmHg	23 (88.5)	15 (88)	8 (88.5)
MDBP awake > 80 mmHg	25 (96)	17 (100)	8 (88.5)
MDBP sleep > 70 mmHg	23 (88.5)	15 (88)	8 (88.5)
MABP awake > 75 mmHg	26 (100)	17 (100)	9 (100)
MABP sleep > 65 mmHg	26 (100)	17 (100)	9 (100)

Variables are given as number (%) or median values. MSBP, Mean systolic blood pressure; MDBP, Mean diastolic blood pressure; MABP, Mean arterial blood pressure.

Table 2: Holter monitoring of overall, awake, and sleep systolic blood pressure, diastolic blood pressure, and mean arterial pressure, before and after stenting in patients with renal artery stenosis

P-value	After stenting	Before stenting	Group	
0.014	178 ± 27	189 ± 26	Overall*	SBP
0.017	178.5 ± 29.9	188.2 ± 26.4	Awake	
0.41	150.8 ± 26.1	157.6 ± 27.6	Sleep	
0.001	97.2 ± 16.2	113.5 ± 24.2	Overall	DBP
0.001	97.6 ± 16.2	135 ± 24	Awake	
0.004	81.7 ± 12.6	91.04 ± 15.2	Sleep	
<0.001	115.8 ± 20	130.6 ± 23.8	Overall	Mean BP
<0.001	115.8 ± 20	130.84 ± 24	Awake	
<0.029	98.2 ± 16.2	106.3 ± 16.6	Sleep	

*Overall blood pressure is a mean of all blood pressure recordings during awake and sleep time by measured by Holter monitoring. SBP, Systolic blood pressure; DBP, Diastolic blood pressure; mean BP, Mean arterial pressure.

Table 3: Holter monitoring of systolic, diastolic, and mean arterial pressure before and after renal artery stenting in patients with and without diabetes

P-value	Without diabetes (n = 16)		P-value	With diabetes (n = 10)		Baseline
	After stent	Before stent		After stent	Before stent	
0.001	176.4 ± 32	187.1 ± 29	0.01	183.2 ± 25.1	192.35 ± 19.2	Overall* SBP
0.001	176.4 ± 32	186.2 ± 30	0.01	182.2 ± 26	191.8 ± 19.8	Awake
0.02	149 ± 29	156.7 ± 27	0.17	154 ± 18	159.6 ± 30	Sleep
0.01	99.3 ± 17	169.9 ± 28	0.06	97.4 ± 14.4	107.5 ± 13	Overall DBP
0.01	99.3 ± 17	116.9 ± 28	0.06	95.4 ± 14	107.5 ± 13.5	Awake
0.05	81 ± 13	90.2 ± 14	0.2	82.8 ± 10	92.4 ± 17	Sleep
0.003	115.6 ± 22	133.3 ± 28	0.02	116.3 ± 16	125.8 ± 12	Overall MAP
0.003	115.5 ± 5	132.9 ± 7	0.02	116.3 ± 17	125.8 ± 12	Awake
0.1	97.5 ± 45	104.2 ± 4	0.03	99.4 ± 13	110.11 ± 17	Sleep

*Overall blood pressure is a mean of all blood pressure recordings during awake and sleep time measured by Holter monitoring.

Table 4: Mean range of serum creatinine improvement before and after renal artery stenting

Improvement >10% from baseline Cr among patients	After stenting	Before stenting	Mean of Cr (mg/dL)	Renal impairment
43.7% (n = 16, P>0.05)	0.19 ± 1.01	0.14 ± 1.06	<1.2	Mild
44.6% (n = 7, P<0.05)	0.6 ± 1.8	0.8 ± 1.8	1.2–3	Moderate
100% (n = 3, P<0.05)	0.2 ± 3.05	0.4 ± 4.2	>3	Severe

intervention: improved mean sCr concentrations were seen in 43.6% of patients with a normal renal function, 44.6% of patients with moderate impairment, and 100% of those with a severely impaired renal function. Patients without diabetes mellitus had higher sCr concentrations at baseline compared with patients with diabetes mellitus (1.58 ± 1.08 mg/dL versus 1.25 ± 0.3 mg/dL; $P < 0.05$). The decrease in sCr was significant in the subgroup of patients without diabetes mellitus (1.58 ± 1.08 to 1.41 ± 0.7 mg/dL; $P < 0.05$), whereas the sCr decrease was not significant in diabetic patients (1.25 ± 0.32 to 1.23 ± 0.38 mg/dL; $P > 0.05$).

Patients with severe nephrosclerosis and diabetes mellitus needed the greatest number of antihypertensive drugs and

had the highest blood pressure levels at baseline. Procedure-related complications occurred in 2 (7.7%) of the 26 cases. The major complications included one femoral artery false aneurysm that was successfully treated with compression, and one patient had acute renal failure.

DISCUSSION

In patients with hypertension and RAS, renal artery angioplasty should ideally provide a cure for hypertension, that is, normal BP without treatment. Angioplasty allows hypertension cure in 50% of patients with fibromuscular RAS, and complications are not frequent in this group. However, the cure rate is lower and the incidence of complications higher among patients with atherosclerotic

RAS.^[13,16] Atherosclerotic patients more frequently suffer from technical failures or subsequent restenosis than those with fibromuscular RAS.^[13,17] They frequently have preexisting primary hypertension, structural changes in large arteries, or an impaired renal function that limit the efficacy and safety of angioplasty.^[2,3,7,13,16] Retrospective series report that the usual BP outcome after angioplasty for atherosclerotic RAS is improvement, that is, a reduction in BP levels and/or in the required number of antihypertensive agents.^[2,13] There are no uniform criteria for assessing the improvement, however, and it may be spontaneous or a consequence of alterations in the drug choice and dosage.^[3,9,13] There is therefore a need for more trials to assess risks and benefits associated with angioplasty in atherosclerotic RAS. The “Scottish and Newcastle Renal Artery Stenosis Collaborative Group” reported in an abstract from a trial of angioplasty versus medical therapy in patients with bilateral or unilateral atherosclerotic RAS.^[10] In the bilateral RAS group ($n = 28$), the drop in systolic BP was significantly greater after angioplasty than after medical therapy, but diastolic BP and creatinine levels did not differ between the two groups after 24 months. In the unilateral RAS group ($n = 27$), there was no significant difference in BP levels after angioplasty or medical therapy. The main outcome variable used was OBP, and no detail was provided concerning treatment standardization.

In our study on percutaneous stent-supported angioplasty of severe atherosclerotic RAS, we observed the stabilization of the renal function and substantial improvement in the blood pressure control at a mean follow-up of 1 month. The observed 11% deterioration of the renal function during follow-up is comparable to previous reports.^[18-39] However, in contrast to Dorros *et al.*'s study,^[24,25,31] which found a deteriorated renal function in 47% of patients with a baseline sCr concentration of >2 mg/dL, and like Zeller *et al.*,^[26] we saw a higher proportion of patients with improved or unchanged sCr among those with the worst renal function at baseline.

The 11% rate of the deteriorated blood pressure control during follow-up was remarkably low, which may be explained by the blood pressure acquisition method in this study. Blood pressure data were exclusively obtained using ambulatory 24-h blood pressure recordings. In our opinion, this type of blood pressure data is more reliable compared to the incidental blood pressure measurements used in all other published studies.^[40-47]

We found an improved renal function that was similar to that reported by van de Ven *et al.*,^[22,28] Iannone *et al.*,^[25] Dorros *et al.*,^[24,25,31] and Zeller *et al.*^[39] The US Multicenter

Registry^[25] demonstrated a significant decrease in the sCr concentration in an unselected study population comparable to ours (1.7 ± 1.1 to 1.3 ± 0.8 mg/dL after 4 years), which is similar to our data.

The present trial was targeted toward patients with unilateral RAS because such cases are more frequent and revascularization is easier, safer, and more likely to result in a favorable BP outcome than in cases with bilateral RAS or RAS affecting a solitary kidney.^[21] Patients with fibromuscular RAS were not included because good evidence is already available that the benefits of angioplasty outweigh the risks in such patients.^[13] It is difficult to differentiate patients with primary hypertension associated with RAS from those having hypertension secondary to RAS, that is, renovascular hypertension.^[1,3] To increase the likelihood of our patients having renovascular hypertension, they were selected on the basis of high-grade stenosis (renal artery diameter reduction 75%) or a stenosis of 60% plus a positive lateralizing test. Patients had been screened for RAS on the basis of poor efficacy and/or tolerance of previous antihypertensive regimen and referred to the participating centers because a unilateral atherosclerotic RAS was present. Although those with refractory hypertension were not included for safety reasons, patients in this trial are representative of the population of cases with unilateral atherosclerotic RAS in whom renal revascularization may be considered.^[1,3] In real life, the early use of a combination of diuretics and angiotensin-converting enzyme inhibitors might have resulted in adequate BP in a larger number of patients. To avoid the biases, poor repeatability, and lack of precision associated with Office Blood Pressure (OBP) determination, therapeutic decisions were based on the average of three measurements, using a semiautomatic device, and the outcome was assessed by 24-h ABP monitoring. Mean ABP levels, including sleep time BP readings, were lower than mean OBP levels, as expected. The difference between ABP and OBP levels was 150/70 mmHg (systolic/diastolic) at randomization in our patients, a difference comparable to that reported at the first visit (120/90 mmHg) in the 50 hypertensive patients analyzed by Bottini *et al.*^[14] The mean ABP levels in the two groups were similar at termination, although the drop in diastolic OBP levels was higher in the control group than in the angioplasty group. These results emphasize the need for an outcome assessment made independent of investigators when blinding is not possible.

Although mean ABP levels were very similar in both groups at termination, angioplasty allowed an easier BP control than medication alone. Treatment scores were higher in the control group than in the angioplasty group,

Table 5: Summary**What is known about this topic**

- The usual BP outcome after angioplasty for atherosclerotic RAS is improvement, that is, a reduction in BP levels and/or in the required number of antihypertensive agents.^[2,13]
- However, the cure rate for hypertension is lower and the incidence of complications higher among patients with atherosclerotic RAS.^[13,16]

What this study adds

- Using a prospective, randomized, open, blinded outcome (PROBE) design, renal angioplasty made BP control easier in the short term but was more frequently associated with complications than conservative management in patients with unilateral atherosclerotic RAS.
- Most patients undergoing angioplasty still needed antihypertensive agents 6 or 12 months after the procedure.

antihypertensive agents being required at termination for all control patients but not for 6 of the 23 allocated to angioplasty (26%). Moreover, 7 of 25 (28%) patients in the control group developed refractory hypertension leading to secondary angioplasty within 6 months. The high BP levels and treatment scores that these seven patients exhibited immediately before secondary angioplasty were included in the analysis. Guidelines for early interruption were established at the design stage of the study and stated that patients should be withdrawn for safety reasons if hypertension were refractory or there were intolerable drug-induced side effects. These guidelines necessitated an on-treatment analysis, with seven patients in the control group having a follow-up of <6 months. We did not perform an intention-to-treat analysis at 6 months because this would have overestimated the drop in BP in the control group, the BP effects of angioplasty being added to those of medication in the seven patients developing refractory hypertension and switching to angioplasty. It is possible that their 6-month ABP levels and treatment score would have been even higher if early termination had not been allowed, raising the possibility that the BP difference between control and angioplasty groups was underestimated because of safety dispositions. The BP effects of the randomized therapeutic regimen, medication, and angioplasty were only compared in the short term, the experimental period lasting for 6 months or less. However, mean OBP levels and the proportion of patients given antihypertensive treatment were similar 1 year after randomization in the control and angioplasty groups, confirming that the BP-lowering effect of angioplasty in the short and medium terms is limited in atherosclerotic RAS. Although the patients were selected on the basis of high-grade stenosis (>75%) and/or a positive lateralizing test, only a minority of them had true renovascular hypertension, that is, a form of hypertension fully reversible after revascularization.^[1,3] In addition to frequently associated primary hypertension and an impaired renal function, individuals with atherosclerotic RAS lose the ability, with increasing age, to reverse the structural vascular changes associated with secondary hypertension.^[45] This underlines the need for the early detection of RAS to allow angioplasty in patients with a short duration of hypertension.^[3,4,23]

The complication rate in our group of patients undergoing angioplasty was substantial (6 of 23, or 26%) and higher than that in many retrospective series.^[13,16] Clinicians involved in the present trial might have used a low threshold to define the presence of a complication. However, they probably applied the same criteria to patients in both treatment groups, and angioplasty was more frequently associated with complications than diagnostic angiography alone. It is also possible that complication rates have been underestimated in some series because they were not documented prospectively in a standardized clinical report form. In the largest retrospective series of angioplasty for atherosclerotic RAS, mechanical complications and acute renal failure (generally reversible) occurred in 26% and 14% of procedures, respectively.^[21] In a prospective randomized trial comparing angioplasty with surgery in atherosclerotic RAS,^[9] there were major and minor complications in 5 and 11 of the 29 patients in the angioplasty group (17% and 48%), respectively. In the present trial, most immediate complications were mild and transient.

CONCLUSION

In summary, previous uncontrolled and unblinded assessments of angioplasty overestimated its potential for lowering BP [Table 5]. Using a prospective, randomized, open, blinded outcome (PROBE) design, we found that angioplasty made the BP control easier in the short term but was more frequently associated with complications than conservative management in patients with unilateral atherosclerotic RAS. Most patients undergoing angioplasty still needed antihypertensive agents 6 or 12 months after the procedure. The reduction in treatment required by patients undergoing angioplasty should therefore be weighed against the risks of complications and restenosis. Previously reported data and this evidence suggest that patients with RAS and little or no renal insufficiency should be offered angioplasty if the underlying disease is fibromuscular dysplasia,^[13] in cases with recurrent pulmonary edema,^[47] and in those with refractory hypertension. Patients with atherosclerotic RAS also have or develop atherosclerotic

plaques or stenosis on extrarenal arteries. In such patients with a stable renal function and controllable hypertension, the effects of angioplasty on the long-term cardiovascular outcome should be compared with those of conservative treatment by using antihypertensive and lipid-lowering agents. Until such a comparison becomes available, the immediate risks and the potential long-term benefits of angioplasty should be weighed for each individual patient, possibly by including patient preference.

REFERENCE

1. Derkx FH, Schalekamp MA. Renal artery stenosis and hypertension. *Lancet* 1994;344:237-9.
2. Rimmer JM, Gennari FJ. Atherosclerotic renovascular disease and progressive renal failure. *Ann Intern Med* 1993;118:712-9.
3. Semple PF, Dominiczak AF. Detection and treatment of renovascular disease: 40 years on. *J Hypertens* 1994;12:729-34.
4. Anderson GH Jr, Blakeman N, Streeten DH. The effect of age on prevalence of secondary forms of hypertension in 4429 consecutively referred patients. *J Hypertens* 1994;12:609-15.
5. Berglund G, Andersson O, Wilhelmson L. Prevalence of primary and secondary hypertension: Studies in a random population sample. *BMJ* 1976;2:554-6.
6. Lewin A, Blaufox MD, Castle H, Entwisle G, Langford H. Apparent prevalence of curable hypertension in the hypertension detection and follow-up program. *Arch Intern Med* 1985;145:424-7.
7. Missouri 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;96:10-4.
8. Davidson RA, Wilcox CS. Newer tests for the diagnosis of renovascular disease. *JAMA* 1992;268:3353-8.
9. Brawn LA, Ramsay LE. Is improvement real with percutaneous transluminal angioplasty in the management of renovascular hypertension? *Lancet* 1987;2:1313-6.
10. "Scottish and Newcastle Renal Artery Stenosis Collaborative Group". Prospective randomized trial of interventional (angioplasty) versus medical therapy in hypertensive patients with atheromatous renal artery stenosis. *J Hypertens* 1994;12:1312.
11. Blum U, Krumme B, Flügel P, Gabelmann A, Lehnert T, Buitrago-Tellez C, *et al.* Treatment of ostial renal-artery stenoses with vascular endoprostheses after unsuccessful balloon angioplasty. *N Engl J Med* 1997;336:459-65.
12. Harden PN, MacLeod MJ, Rodger RS, Baxter GM, Connell JM, Dominiczak AF, *et al.* Effect of renal-artery stenting on progression of renovascular renal failure. *Lancet* 1997;349:1133-6.
13. Ramsay LE, Waller PC. Blood pressure response to percutaneous transluminal angioplasty for renovascular hypertension: An overview of published series. *BMJ* 1990;300:569-72.
14. Bottini PB, Carr AA, Rhoades RB, Prisant LM. Variability of indirect methods used to determine blood pressure: Office vs mean 24-hour automated blood pressures. *Arch Intern Med* 1992;152:139-44.
15. Cockcroft DW, Gault H. Prediction of creatinine clearance from serum creatinine. *Nephron* 1976;16:31-41.
16. Malher F, Triller J, Weidmann P, Nachbur B. Complications in percutaneous transluminal dilatation of renal arteries. *Nephron* 1986;44:60-3.
17. Plouin PF, Darné B, Chatellier G, Pannier I, Battaglia C, Raynaud A, *et al.* Restenosis after a first percutaneous transluminal renal angioplasty. *Hypertension* 1993;21:89-96.
18. Blum U, Krumme B, Flügel P, Gabelmann A, Lehnert T, Buitrago-Tellez C, *et al.* Treatment of ostial renal-artery stenoses with vascular endoprostheses after unsuccessful balloon angioplasty. *N Engl J Med* 1997;336:459-65.
19. Taylor A, Sheppard D, MacLeod MJ, Harden P, Baxter GM, Edwards RD, *et al.* Renal artery stent placement in renal artery stenosis: Technical and early clinical results. *Clin Radiol* 1997;52:451-7.
20. Henry M, Amor M, Henry I, Ethevenot G, Allaoui M, Tricoche O, *et al.* Stent placement in the renal artery: Three-year experience with the Palmaz stent. *J Vasc Interv Radiol* 1996;7:343-50.
21. Harden PN, MacLeod MJ, Rodger RS, Baxter GM, Connell JM, Dominiczak AF, *et al.* Effect of renal-artery stenting on progression of renovascular renal failure. *Lancet* 1997;349:1133-6.
22. van de Ven PJ, Beutler JJ, Kaatee R, Beek FJ, Mali WP, Geyskes GG, *et al.* Transluminal vascular stent for ostial atherosclerotic renal artery stenosis. *Lancet* 1995;346:672-4.
23. 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.
24. Dorros G, Prince C, Mathiak L. Stenting of renal artery stenosis achieves better relief of the obstructive lesion than balloon angioplasty. *Cathet Cardiovasc Diagn* 1993;29:191-8.
25. Dorros G, Jaff M, Mathiak L, He T; Multicenter Registry Participants. Multicenter Palmaz stent renal artery stenosis revascularization registry report: Four-year follow-up of 1058 successful patients. *Catheter Cardiovasc Interv* 2002;55:182-8.
26. Fiala LA, Jackson MR, Gillespie DL, O'Donnell SD, Lukens M, Gorman P. Primary stenting of atherosclerotic renal artery ostial stenosis. *Ann Vasc Surg* 1998;12:128-33.
27. Plouin PF, Chatellier G, Darne B, Raynaud A. Blood pressure outcome of angioplasty in atherosclerotic renal artery stenosis: A randomized trial. *Essai Multicentrique Medicaments vs Angioplastie Study Group. Hypertension* 1998;31:823-9.
28. Van de Ven PJ, Kaatee R, Beutler JJ, Beek FJ, Woittiez AJ, Buskens E, *et al.* Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: A randomised trial. *Lancet* 1999;353:282-6.
29. White CJ, Ramee SR, Collins TJ, Jenkins JS, Escobar A, Shaw D. Renal artery stent placement: Utility in lesions difficult to treat with balloon angioplasty. *J Am Coll Cardiol* 1999;30:1445-50.
30. Radermacher J, Chavan A, Bleck J, Vitzthum A, Stoess B, Gebel MJ, *et al.* Use of Doppler ultrasonography to predict the outcome of therapy for renal-artery stenosis. *N Engl J Med* 2001;344:410-7.
31. Dorros G, Jaff M, Mathiak L, Dorros II, Lowe A, Murphy K, *et al.* Four-year follow-up of Palmaz-Schatz stent revascularization as treatment for atherosclerotic renal artery stenosis. *Circulation* 1998;98:642-7.
32. Shannon HM, Gillespie IN, Moss JG. Salvage of the solitary kidney by insertion of a renal artery stent. *AJR Am J Roentgenol* 1998;171:217-22.
33. Paulsen D, Klow NE, Rogstad B, Leivestad T, Lien B, Vatne K, *et al.* Preservation of renal function by percutaneous transluminal angioplasty in ischemic renal disease. *Nephrol Dial Transplant* 1999;14:1454-61.
34. Isles CG, Robertson S, Hill D. Management of renovascular disease: A review of renal artery stenting in ten studies. *QJM* 1999;92:159-67.
35. Watson PS, Hadjipetrou P, Cox SV, Piemonte TC, Eisenhauer AC. Effect of renal artery stenting on renal function and size in patients with atherosclerotic renovascular disease. *Circulation* 2000;102:1671-7.
36. Beutler JJ, Van Ampting JM, Van De Ven PJ, Koomans HA, Beek FJ, Woittiez AJ, *et al.* Long-term effects of arterial stenting on kidney function for patients with ostial atherosclerotic renal artery stenosis and renal insufficiency. *J Am Soc Nephrol* 2001;12:1475-81.
37. Muray S, Martin M, Amoedo ML, García C, Jornet AR, Vera M, *et al.* Rapid decline in renal function reflects reversibility and predicts the outcome after angioplasty in renal artery stenosis. *Am J Kidney Dis* 2002;39:60-6.
38. Rocha-Singh KJ, Ahuja RK, Sung CH, Rutherford J. Long-term renal function preservation after renal artery stenting in patients with progressive ischemic nephropathy. *Catheter Cardiovasc Interv* 2002;57:135-41.
39. Zeller T. Endovascular treatment of renal artery stenosis: Technical aspects, long-term clinical results, and restenosis-rate. In: Marco J, Serruys P, Biamino G, *et al.*, editors. *The Paris Course on Revascularization* 2001. p. 419-50.
40. Weibull H, Bergqvist D, Bergentz SE, Jonsson K, Hulthén UL, Manhem P. Percutaneous transluminal renal angioplasty versus surgical reconstruction

- of atherosclerotic renal artery stenosis: A prospective randomized study. *J Vasc Surg* 1993;18:841-52.
41. Merlo J, Wessling A, Melander A. Comparison of dose standard units for drug utilisation studies. *Eur J Clin Pharmacol* 1996;50:27-30.
 42. Jamieson MJ, Webster J, Witte K, Huggins MM, MacDonald TM, De Beaux A, *et al.* An evaluation of the A and D UA-751 semi-automated cuff-oscillometric sphygmomanometer. *J Hypertens* 1990;8:377-81.
 43. Canzanello VJ, Millan VG, Spiegel JE, Ponce SP, Kopelman RI, Madias NE. Percutaneous transluminal renal angioplasty in management of atherosclerotic renovascular hypertension: results in 100 patients. *Hypertension* 1989;13:163-72.
 44. Miyamori I, Yasuhara S, Matsubara T, Takasaki H, Takeda R. Comparative effects of captopril and nifedipine on split renal function in renovascular hypertension. *Am J Hypertens* 1988;1:359-63.
 45. Streeten DH, Anderson GH, Wagner S. Effect of age on response of secondary hypertension to specific treatment. *Am J Hypertens* 1990;3:360-5.
 46. Abuelo JG. Diagnosing vascular causes of renal failure. *Ann Intern Med* 1995;123:601-14.
 47. Pickering TG, Devereux RB, James GD, Silane MF, Herman L, Sotelo JE, *et al.* Recurrent pulmonary edema in hypertension due to bilateral renal artery stenosis: treatment by angioplasty or surgical revascularization. *Lancet* 1988;2:551-2.

Source of Support: Nil, **Conflict of Interest:** None declared.

Staying in touch with the journal

1) Table of Contents (TOC) email alert

Receive an email alert containing the TOC when a new complete issue of the journal is made available online. To register for TOC alerts go to www.jcdonline.com/signup.asp.

2) RSS feeds

Really Simple Syndication (RSS) helps you to get alerts on new publication right on your desktop without going to the journal's website. You need a software (e.g. RSSReader, Feed Demon, FeedReader, My Yahoo!, NewsGator and NewzCrawler) to get advantage of this tool. RSS feeds can also be read through FireFox or Microsoft Outlook 2007. Once any of these small (and mostly free) software is installed, add www.jcdonline.com/rssfeed.asp as one of the feeds.