

TREATMENT OF OSTIAL RENAL-ARTERY STENOSES WITH VASCULAR ENDOPROSTHESES AFTER UNSUCCESSFUL BALLOON ANGIOPLASTY

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ABSTRACT

Background Percutaneous transluminal renal angioplasty is a safe and effective treatment for non-ostial stenoses of the renal arteries, but it has proved to be disappointing for ostial stenoses. Therefore, we prospectively studied the use of intravascular stents for the treatment of critical ostial stenoses after unsuccessful balloon angioplasty.

Methods Stainless-steel endoprostheses were placed across 74 renal-artery stenoses located within 5 mm of the aortic lumen in 68 patients with hypertension. Twenty patients had mild or severe renal dysfunction. The indications for stent placement were elastic recoil (63 arteries) or dissection (1 artery) of the vessel after angioplasty, or restenosis after initially successful balloon angioplasty (10 arteries). Patients were followed for a mean of 27 months with measurements of blood pressure and serum creatinine, duplex sonography, and intraarterial angiography.

Results Initial technical success was achieved in all patients. Minor complications (local hematomas) occurred in only three patients; there were no major complications. Eighty-four percent of the patients were free of primary occlusion 60 months after the procedure. Restenosis of more than 50 percent of the vessel diameter occurred in 8 of 74 arteries (11 percent). Reintervention resulted in a secondary patency rate of 92 percent. Long-term normalization of blood pressure was achieved in 11 patients (16 percent). Serum creatinine levels did not change significantly after successful stent implantation in patients with previously impaired renal function.

Conclusions Accurate placement of renal-artery stents is technically feasible without major complications. The favorable early and long-term results suggest that primary stent placement is an effective treatment for renal-artery stenosis involving the ostium. (N Engl J Med 1997;336:459-65.)

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RENAL-ARTERY stenosis is the most common cause of secondary hypertension, with a prevalence of about 1 percent in the general population of people with hypertension.^{1,2} Severe arterial stenosis may also lead to inadequate renal plasma flow and impair the excretory function of the kidney.^{3,4}

The technical and functional results of conventional balloon angioplasty of nonostial renal-artery stenoses caused by fibromuscular dysplasia⁵⁻⁷ or atherosclerosis have been reported extensively.⁷⁻¹¹ In pa-

tients with obstruction of inflow to the renal artery due to an aortic plaque, however, the results of balloon angioplasty have been disappointing. The initial success rates range from 24 to 35 percent,¹⁰⁻¹³ and the rates of recurrence of the lesions from 15 to 42 percent.¹⁴⁻¹⁶ Therefore, this type of renovascular disease is commonly treated by primary surgical intervention.^{13,17} To overcome the problem of elastic recoil after angioplasty, recent reports recommend different types of intravascular stents for the treatment of nonostial renal-artery stenoses.¹⁸⁻²⁵ Few data are available on the use of intravascular stents for the critical ostial lesions.^{20,23-25}

We present long-term clinical, duplex sonographic, and angiographic results of the treatment of ostial renal-artery atheroma with vascular endoprostheses.

METHODS**Subjects**

From March 1989 through March 1996, we treated 82 arteries in 75 patients with conventional balloon angioplasty for atherosclerotic lesions involving the orifice of the renal arteries, with complete technical success in 10 percent. As adjunctive therapy, short endoprostheses (Palmaz stent, Johnson and Johnson Interventional Systems, Warren, N.J.) were implanted in 74 arteries of 68 patients (44 men and 24 women). Six patients had severe contralateral stenosis, and six patients had a single functioning kidney. Ostial lesions were defined as stenoses of more than 50 percent of the diameter of the renal artery within 5 mm of the aortic lumen, caused by atherosclerotic disease of the aorta (Fig. 1A).^{12,26} The degree of stenosis was determined by the reduction in the luminal diameter. All patients had a history of sustained hypertension resistant to intensive antihypertensive treatment. During the study period, no patients underwent primary surgical revascularization or required surgery after failed balloon angioplasty or stent placement. The base-line characteristics of the patients are shown in Table 1.

The indications for stent placement were unsuccessful balloon angioplasty with elastic recoil immediately after intervention, residual stenosis greater than 50 percent, and a transstenotic gradient greater than 20 mm Hg (63 arteries); restenosis (10 arteries); or dissection with an obstructing intimal flap (1 artery). All the patients gave written informed consent.

Preintervention Diagnostic Workup

The diagnosis of renal-artery stenosis was based on color duplex sonography, intraarterial angiography, and a transstenotic pressure gradient greater than 20 mm Hg.

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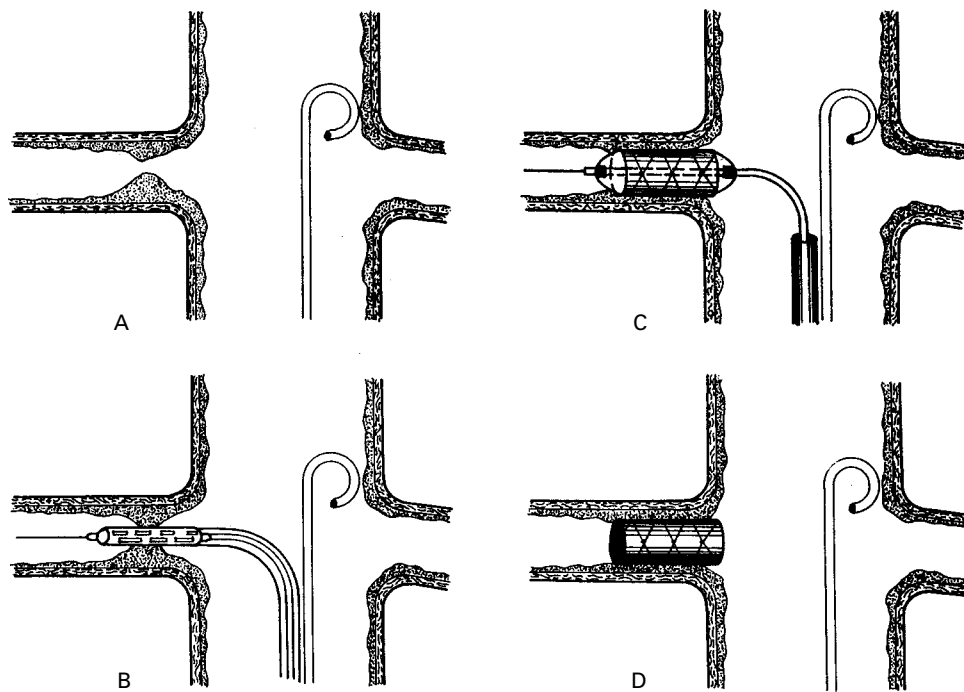


Figure 1. Schematic Presentation of the Atherosclerotic Ostial Renal-Artery Lesion and the Technique of Stent Placement. In Panel A, atherosclerotic aortic plaque extends into the orifice of the renal artery, compromising blood flow. After predilatation, the guiding catheter–balloon–stent assembly is placed across the lesion (Panel B). After removal of the delivery system, the stent is adjusted to protrude 1 to 2 mm into the aortic lumen, thus completely covering the ostial lesion, and then balloon-expanded (Panel C). The endoprosthesis is left in place, completely covering the atheromatous lesion, after the removal of the guide wire and balloon catheter (Panel D).

All duplex sonographic studies were performed by the same two experienced investigators before intraarterial angiography. The patients were examined in the supine and lateral decubitus positions with a 2.5-to-3.5-MHz phased-array transducer (128/XP 10, Acuson, Mountain View, Calif.). After conventional sonography for determination of the size of the kidney and evaluation of parenchymal disorders, the color mode was added for vascular imaging.

The main renal artery and the segmental and interlobular arteries of the upper pole, midportion, and lower pole of the kidney were visualized by the color mode. In each instance, the angle-corrected (<60 degrees) peak systolic and end-diastolic velocities were determined from the Doppler spectra, and the resistive index was calculated. The resistive-index values of six different spectral samples obtained in the intrarenal arteries were averaged to the mean resistive index of the kidney, which was used for evaluation. In each patient, the difference between the intrarenal resistive indexes of the two kidneys was determined.^{27,28}

The criteria for confirmed stenosis were an angle-corrected peak systolic velocity of more than 2 m per second in the main renal artery²⁹ and a difference of more than 0.05 between the resistive indexes, with the smaller index on the stenotic side.^{27,28}

Immediately before angioplasty or stent placement, multiplane abdominal aortograms (anterior–posterior, left anterior oblique 30-degree, and right anterior oblique 30-degree projections) were obtained to define the anatomy of the obstruction. A computer program with an edge-detecting algorithm (Siemens, Erlangen, Germany) was used to calculate the diameter of the renal artery. This diameter was 4 mm in 3 arteries, 5 mm in 34 arteries, 6 mm in 33 arteries, and 7 mm in 4 arteries. The degree of stenosis was calculated as 1 minus the ratio of the diameter of the lumen at

the stenosis to the diameter of the lumen of the uninvolved renal artery distal to the stenosis; these values were then expressed as percentages.

The renal arterial pressure proximal and distal to the lesion (transstenotic pressure gradient) was determined before and after balloon angioplasty, as well as before reintervention in cases of restenosis, with use of a 5-French end-hole catheter.

Technique of Angioplasty and Stent Implantation

The principles and technical aspects of balloon angioplasty have been described elsewhere.³⁰ In all our patients, the lesion was dilated with a 4.8-French angioplasty balloon catheter (Olbert catheter, Meadox Surgimed, Stenlose, Denmark) passed through a valved 8-French introducer sheath with a femoral approach before placement of the stent. To evaluate the immediate technical result after angioplasty and to position the stent precisely, we introduced an aortic catheter through a contralateral femoral artery.

In cases of unsatisfactory angioplasty results, as assessed by repeated angiography and determination of the transstenotic pressure gradient, we immediately implanted an intravascular stent over a stiff 0.5-mm (0.020 in.) guide wire that was left in the renal artery. To cover the atheromatous lesion, we used the short Palmaz endoprosthesis in all patients. The stent was a stainless-steel tube 10 mm (51 arteries) or 15 mm (23 arteries) in length, which was crimped onto the same angioplasty balloon used for the previous angioplasty. With a hockey-stick-shaped device (Medtronic, Interventional Vascular, Danvers, Mass.), the guiding catheter–balloon–stent assembly was then passed over the guide wire across the lesion (Fig. 1B). The delivery system was then with-

TABLE 1. CHARACTERISTICS OF 68 PATIENTS WITH OSTIAL RENAL-ARTERY STENOSES.

| CHARACTERISTIC | VALUE* | RANGE |
|---|-----------|---------|
| Sex (M/F) | 44/24 | |
| Age (yr) | 60.1±10.0 | 31–80 |
| Blood pressure (mm Hg) | | |
| Mean | 133±15 | 106–170 |
| Systolic | 188±28 | 150–250 |
| Diastolic | 105±11 | 90–130 |
| Serum creatinine (mg/dl) | 1.23±0.60 | 0.5–3.9 |
| Antihypertensive drugs (no.)† | 2.9±0.9 | 1–5 |
| Coexisting risk factors (no. of patients) | | |
| Smoking | 28 | |
| Dyslipidemia | 21 | |
| Diabetes mellitus type II | 4 | |

*Plus-minus values are means ±SD.

†Antihypertensive drugs included diuretics, beta-blockers, calcium-channel blockers, angiotensin-converting-enzyme inhibitors, and alpha-blockers.

drawn into the aorta, leaving the stent in place mounted on the balloon (Fig. 1C). For accurate positioning of the endoprosthesis, angiography was performed with the catheter introduced contralaterally to adjust the position of the stent. The endoprosthesis was fitted to protrude 1 to 2 mm into the aortic lumen to cover the aortic plaque completely. The balloon was then inflated and the stent was expanded to a diameter of 1 to 1.2 times that of the renal artery (4 to 7 mm). The balloon was then removed (Fig. 1D), and post-procedural angiography was performed.

A bolus dose of heparin (5000 IU) was administered intravenously during the procedure, and the infusion was then continued for two days at a dose of 20,000 to 30,000 IU per day after the removal of the introducer sheath to achieve a prolongation of the partial-thromboplastin time to 60 seconds. Antiplatelet medication (100 mg of aspirin per day, or, when adverse effects occurred, 250 mg of ticlopidine per day) was given.

Follow-up Protocol

Follow-up studies included duplex sonography, angiography, and monitoring of blood pressure, drug therapy, and serum creatinine. These follow-up measurements, except for angiography, were performed before discharge, at 3, 6, and 12 months, and then every year on an outpatient basis. Angiography was performed when restenosis was suspected on the basis of clinical findings or duplex sonography. Regular transbrachial intraarterial angiographic reevaluation was performed at 12 and 24 months. Five of the 68 patients were not available for angiographic follow-up studies.

Definition of Technical and Functional Results

Complete technical success after angioplasty was defined as residual stenosis of less than 50 percent according to angiography and a transstenotic pressure gradient of less than 20 mm Hg.

Restenosis was defined according to color duplex sonography as a peak systolic velocity of more than 2 m per second and a difference of more than 0.05 between resistive indexes.²⁷ All restenoses suspected on the basis of sonography were evaluated by intraarterial angiography and determination of the pressure gradient. Restenosis was defined angiographically as the development of stenosis of more than 50 percent of the luminal diameter and a transstenotic pressure gradient of more than 20 mm Hg.

The stent was considered to have produced primary patency if no other procedure had to be performed. Secondary patency was

considered to have been achieved if dilatation within the stent or additional stent implantation was necessary at reintervention.

Blood pressure, antihypertensive treatment, and serum creatinine concentrations were monitored before the intervention and during follow-up. The rate of clinical benefit was assessed during the hospital stay and at the first follow-up examination according to the criteria of the Cooperative Study of Renovascular Hypertension.³¹ Reversal of hypertension corresponded to a diastolic pressure of 90 mm Hg or less and no need for medication. Improvement corresponded to a diastolic pressure of 91 to 109 mm Hg and a decrease of at least 15 percent, or a diastolic pressure of 91 to 109 mm Hg, a decrease of at least 10 percent, and withdrawal of at least one drug from the treatment regimen.

A serum creatinine level higher than 1.4 mg per deciliter (124 μ mol per liter) was considered abnormal and indicative of renal dysfunction (mild dysfunction, 1.5 to 1.9 mg per deciliter [134 to 169 μ mol per liter]; severe dysfunction, 2.0 mg per deciliter [177 μ mol per liter]).

Statistical Analysis

All values are given as means ±SD or as numbers of patients and percentages. To determine the statistical significance of the differences between the pre- and post-procedural values for serum creatinine and arterial blood pressure, we applied the Mann-Whitney rank-sum test. A P value of less than 0.05 was considered to indicate statistical significance. Cumulative occlusion-free survival rates were obtained with the Kaplan-Meier method.

RESULTS

Follow-up

The mean follow-up period for the total study group was 27 months (range, 3 to 84). Seven patients were followed for 60 months, 13 for 48 months, 16 for 36 months, 27 for 24 months, and 47 for 12 months. Three patients died 8, 19, and 35 months after the procedure of diseases unrelated to revascularization (obstructive lung disease, myocardial infarction, and lung carcinoma, respectively).

Stent Implantation

A total of 74 endoprostheses were implanted in 68 patients. After unsatisfactory conventional angioplasty, all stenting procedures were technically successful. In 71 of the 74 arteries, the deployed stent projected about 1 to 2 mm into the aortic lumen, thus covering the aortic plaque at the orifice of the renal artery (Fig. 2A and 2B). In 3 of the 74 arteries, the stent protruded 3 to 4 mm into the aorta without causing any problem. Because of slightly inaccurate placement of the original endoprosthesis, a second overlapping 10-mm Palmaz stent had to be implanted in two arteries to cover the atheromatous lesion or the ostium completely. In six patients, there was severe contralateral stenosis, leading to the consecutive placement of bilateral stents during the same procedure. At post-procedural angiography, no residual stenosis could be demonstrated.

There were no major complications. In three patients, a local hematoma that did not require further intervention was observed at the puncture site. The average hospital stay was 4.5±2.4 days (range, 2 to 12).



A



B

Figure 2. Ostial Renal-Artery Stenosis in a 49-Year-Old Patient with Severe Hypertension Treated Endoluminally with a 15-mm-Long Palmaz Endoprosthesis.

In Panel A, preinterventional angiography demonstrates high-grade ostial stenosis of the right renal artery. In Panel B, angiography 24 months after the procedure shows the correctly placed endoprosthesis with the proximal edge protruding into the aortic lumen without residual stenosis (arrowheads).

Effects on Blood Pressure and Renal Function

Follow-up data on blood pressure and renal function were available for all patients. Reversal of hypertension was achieved in 11 patients (16 percent) after successful renal-artery stenting. In 42 patients (62 percent) hypertension was classified as improved, and in the remaining 15 (22 percent) as unchanged. No late changes in blood pressure were observed more than three months after the initial procedure. The overall mean changes in systolic and diastolic blood pressure are shown in Figure 3.

Renal function, as indicated by serum creatinine levels, was stable in all the patients, with no significant change during follow-up (Fig. 4). Serum creatinine levels also remained unchanged immediately after the procedure and during follow-up in the subgroup of patients with mild (17 patients) or severe (3 patients) renal impairment.

Color Duplex Sonography

A total of 349 color sonographic examinations were performed in the 68 patients to monitor renal-artery stenosis before and after the intervention. The presence of a stent did not affect the ability to record a Doppler signal along the endoprosthesis and the renal artery. The mean peak systolic velocity in the main renal artery decreased from 377 ± 104 cm per second before stent implantation to 130 ± 45 cm per second after stent implantation ($P < 0.001$). The intrarenal resistive index of the stenotic kidneys increased from 0.62 ± 0.09 to 0.71 ± 0.08 ($P < 0.001$), and the mean difference between the resistive indexes decreased from 0.09 ± 0.06 to 0.02 ± 0.02 ($P < 0.001$) within the first week after the procedure. During long-term follow-up, acceleration of peak systolic velocity (>2 m per second) was found in 16 patients. However, only 8 of the 16 patients presented with significant differences in resistive index (>0.05) between the two kidneys. Angiography confirmed substantial restenoses (>50 percent) in all eight patients. In the other eight patients with accelerated peak systolic velocities of the main renal artery, angiography revealed only slight intimal hyperplasia without substantial restenosis.

Substantial restenoses were diagnosed by duplex sonography in two of the eight patients after 3 months, in three patients after 6 months, in two patients after 12 months, and in one patient after 24 months.

Follow-up Angiography, Restenosis, and Reintervention

Repeated intraarterial angiography was performed 12 and 24 months after the initial intervention. In 46 of 48 arteries, angiography at 12 months revealed a thin, smooth intimal layer that covered the stent struts and the spaces between them without causing substantial restenosis (Fig. 2B). In two renal

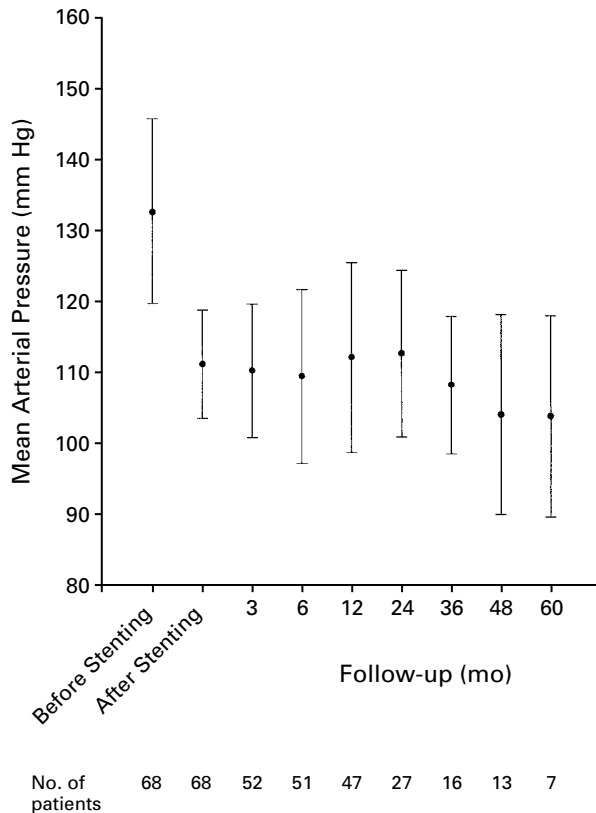


Figure 3. Mean (\pm SD) Arterial Pressure in 68 Patients Treated for Ostial Renal-Artery Stenoses. All postimplantation values are significantly lower than baseline values ($P < 0.001$).

arteries, however, restenosis was detected. Angiography of 28 arteries at 24 months found recurrent stenosis greater than 50 percent in one more patient. In all other patients, angiography at 24 months showed no change from the results at 12 months along the stent tract.

Restenosis occurred at the proximal edge of the endoprosthesis in four arteries and between the proximal part and the midportion in four arteries. The original diameter of the implanted endoprosthesis was 4 mm in one artery, 5 mm in four arteries, and 6 mm in three arteries. In all but one instance, the restenotic lesion was irregularly shaped and not concentric, suggesting the presence of organizing thrombotic material.

Reintervention was performed in six of the eight patients who had restenosis during follow-up. For restenosis, angioplasty with enlargement of the original diameter of the renal artery was performed successfully in the six patients, and it was followed by implantation of a second short stent in all of them. The remaining two patients with restenosis, who had normal renal function and unchanged hyperten-

sion after the initial stenting procedure, did not undergo reintervention. Hypertension was controlled by medication.

The cumulative primary and secondary occlusion-free survival rates are shown in Figure 5.

DISCUSSION

Our prospective, long-term study found that endoluminal treatment of ostial stenoses of the renal arteries is a safe and effective alternative to surgery. With a guiding catheter across the stenotic lesion and a control catheter from a contralateral point of access, the endoprosthesis can be placed accurately and without major complications.

Restenosis after stent implantation is usually caused by myointimal hyperplasia. In most of our patients, a minor deposition of tissue without substantial restenosis, angiographically visualized as a thin, smooth layer over the stent struts and the spaces between them, was observed after 12 and 24 months. This phenomenon is a result of the normal healing process in patients with vascular stents, with an initial thrombotic layer covering the stent struts and its progressive replacement first by fibromuscular tissue and later by collagen.³² In 8 of 74 renal arteries, however, restenosis along the implanted endoprostheses was diagnosed by color duplex sonography. In seven of these eight arteries, subsequent angiography demonstrated irregularly shaped luminal narrowing located at the proximal portion or the midportion of the stent, suggesting thrombus formation rather than intimal hyperplasia. In these patients, the stent may have been deployed along the atheromatous plaque without proper embedding, thus causing thrombosis over the stented surface. In addition, flow separation and higher shear rates resulting from unfavorable volume-surface relations of the blood circulating through the stent may have been responsible for restenosis.³³ Restenosis caused by disturbed flow and shear stress may be particularly likely in renal lesions because of the angle of departure from the aorta.²⁰

The frequency of restenosis during long-term follow-up was 10 percent, similar to the 16 percent reported during short-term follow-up by MacLeod et al.,²³ van de Ven et al.,²⁴ and Henry et al.²⁵ Our restenosis rate was not significantly affected by vessel diameter and was considerably lower than those in other studies that used longer stents (20 to 39 percent).²⁰⁻²² Our data, therefore, suggest that the most important factor promoting long-term patency is the short length of the endoprosthesis, which ensures a rapid, nondisturbed arterial flow. In addition, the implantation technique we used, with complete covering of the orifice of the renal artery and slight enlargement of the diameter of the original vessel, may be responsible for the favorable outcome.

Repeated angioplasty and placement of an addi-

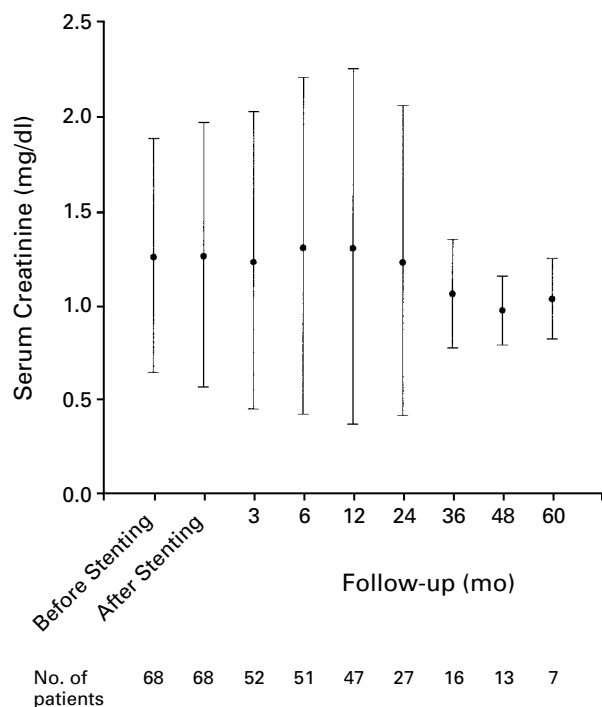


Figure 4. Mean (\pm SD) Serum Creatinine Levels in 68 Patients Treated for Ostial Renal-Artery Stenoses.

Postimplantation values are not significantly different from base-line values ($P > 0.05$). To convert values for serum creatinine to micromoles per liter, multiply by 88.4.

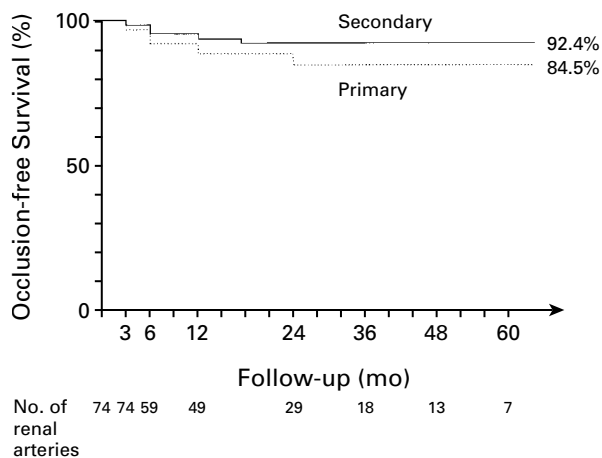


Figure 5. Cumulative Primary and Secondary Occlusion-free Survival Rates after Stent Placement in 74 Ostial Renal-Artery Stenoses in 68 Patients.

tional, overlapping, short stent in patients with recurrent stenosis resulted in a cumulative rate of secondary patency of 92 percent at 60 months. These results are substantially better than those when balloon angioplasty alone is used for ostial lesions.¹⁰⁻¹⁶

The long-term outcome in our patients, with reversal of hypertension in 16 percent and improvement in 62 percent, is similar to the results reported in previous studies.³⁴ Renal function in the 20 patients who had mild or severe renal dysfunction before the intervention did not change during follow-up. This finding is clinically important, because untreated stenosis may progress in severity, resulting in renal-artery occlusion, loss of renal mass, and a subsequent decrease in kidney function.^{35,36}

With respect to primary patency, the technical success rate of surgical revascularization is similar to that of endoluminal treatment with stent implantation. However, surgical revascularization is associated with a perioperative mortality rate of 2 to 7 percent, morbidity in 17 to 31 percent of patients, and deterioration of renal function in 11 to 31 percent of patients.³⁷⁻⁴⁰ The rate of reocclusion or restenosis after surgical renal-artery repair is 3 to 4 percent.^{37,40}

Although our study used historical controls and was not randomized, the extremely low complication rate, low procedure costs, and overall therapeutic value as compared with that of well-performed vascular surgical procedures strongly suggest that endoluminal therapy with short stents is safe and clinically effective in patients with ostial renovascular disease. A prospective, randomized study comparing this strategy with surgery now seems indicated.

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REFERENCES

- Mann SJ, Pickering TG. Detection of renovascular hypertension: state of the art. *Ann Intern Med* 1992;117:845-53.
- Derx FHM, Schalekamp MADH. Renal artery stenosis and hypertension. *Lancet* 1994;344:237-9.
- Dean RH, Tribble RW, Hansen KJ, O'Neil E, Craven TE, Redding JF II. Evolution of renal insufficiency in ischemic nephropathy. *Ann Surg* 1991;213:446-55.
- Hansen KJ. Prevalence of ischemic nephropathy in the atherosclerotic population. *Am J Kidney Dis* 1994;24:615-21.
- Millan VG, McCauley J, Kopelman RI, Madias NE. Percutaneous transluminal renal angioplasty in nonatherosclerotic renovascular hypertension: long-term results. *Hypertension* 1985;7:668-74.
- Tegtmeyer CJ, Selby JB, Hartwell GD, Ayers C, Tegtmeyer V. Results and complications of angioplasty in fibromuscular disease. *Circulation* 1991;83:Suppl 2:1155-61.
- Sos TA, Pickering TG, Sniderman K, et al. Percutaneous transluminal renal angioplasty in renovascular hypertension due to atheroma or fibromuscular dysplasia. *N Engl J Med* 1983;309:274-9.
- Tegtmeyer CJ, Kellum CD, Ayers C. Percutaneous transluminal angioplasty of the renal artery: results and long-term follow-up. *Radiology* 1984;153:77-84.
- Martin LG, Casarella WJ, Alspaugh JP, Chuang VP. Renal artery angioplasty: increased technical success and decreased complications in the second 100 patients. *Radiology* 1986;159:631-4.
- Canzanello VJ, Millan VG, Spiegel JE, Ponce SP, Kopelman RI, Ma-

- dias NE. Percutaneous transluminal renal angioplasty in management of atherosclerotic renovascular hypertension: results in 100 patients. *Hypertension* 1989;13:163-72.
11. Martin LG, Cork RD, Kaufman SL. Long-term results of angioplasty in 110 patients with renal artery stenosis. *J Vasc Interv Radiol* 1992;3:619-26.
 12. Cicuto KP, McLean GK, Oleaga JA, Freiman DB, Grossman RA, Ring EJ. Renal artery stenosis: anatomic classification for percutaneous transluminal angioplasty. *AJR Am J Roentgenol* 1981;137:599-601.
 13. Eldrup-Jorgensen J, Harvey HR, Sampson LN, Amberson SM, Bredenberg CE. Should percutaneous transluminal renal artery angioplasty be applied to ostial artery atherosclerosis? *J Vasc Surg* 1995;21:909-15.
 14. Kremer-Hovinga TK, de Jong PE, de Zeeuw D, Donker AJM, Schuur KH, van der Hem GK. Restenosis prevalence and long-term effects on renal function after percutaneous transluminal renal angioplasty. *Nephron* 1986;44:Suppl 1:64-7.
 15. Plouin P-F, Darne B, Chatellier G, et al. Restenosis after a first percutaneous transluminal renal angioplasty. *Hypertension* 1993;21:89-96.
 16. Jensen G, Zachrisson B-F, Delin K, Volkman R, Aurell M. Treatment of renovascular hypertension: one year results of renal angioplasty. *Kidney Int* 1995;48:1936-45.
 17. Dean RH, Callis JT, Smith BM, Meacham PW. Failed percutaneous transluminal angioplasty: experience with lesions requiring operative intervention. *J Vasc Surg* 1987;6:301-7.
 18. Wilms GE, Peene PT, Baert AL, et al. Renal artery stent placement with use of the Wallstent endoprosthesis. *Radiology* 1991;179:457-62.
 19. Kuhn FP, Kutkuhn B, Torsello G, Mödder U. Renal artery stenosis: preliminary results of treatment with the Strecker stent. *Radiology* 1991;180:367-72.
 20. Rees CR, Palmaz JC, Becker GJ, et al. Palmaz stent in atherosclerotic stenoses involving the ostia of the renal arteries: preliminary report of a multicenter study. *Radiology* 1991;181:507-14.
 21. Hennequin LH, Joffre FG, Rousseau HP, et al. Renal artery stent placement: long-term results with the Wallstent endoprosthesis. *Radiology* 1994;191:713-9.
 22. 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;75:1051-5.
 23. MacLeod M, Taylor AD, Baxter G, et al. Renal artery stenosis managed by Palmaz stent insertion: technical and clinical outcome. *J Hypertens* 1995;13:1791-5.
 24. van de Ven PJG, Beutler JJ, Kaatee R, et al. Transluminal vascular stent for ostial atherosclerotic renal artery stenosis. *Lancet* 1995;346:672-4.
 25. Henry M, Amor M, Henry I, et al. Stent placement in the renal artery: three-year experience with the Palmaz stent. *J Vasc Interv Radiol* 1996;7:343-50.
 26. Kaatee R, Beek FJA, Verschuyf EJ, et al. Atherosclerotic renal artery stenosis: ostial or truncal? *Radiology* 1996;199:637-40.
 27. Krumme B, Blum U, Schwertfeger E, et al. Diagnosis of renovascular disease by intra- and extrarenal Doppler scanning. *Kidney Int* 1996;50:1288-92.
 28. Schwerek WB, Restrepo IK, Stellwaag M, Klose KJ, Schade-Brittinger C. Renal artery stenosis: grading with image-directed Doppler US evaluation of renal resistive index. *Radiology* 1994;190:785-90.
 29. Olin JW, Piedmonte MR, Young JR, DeAnna S, Grubb M, Childs MB. The utility of duplex ultrasound scanning of the renal arteries for diagnosing significant renal artery stenosis. *Ann Intern Med* 1995;122:833-8.
 30. Tegtmeyer CJ, Sos TA. Techniques of renal angioplasty. *Radiology* 1986;161:577-86.
 31. Standards of Practice Committee of the Society of Cardiovascular and Interventional Radiology. Guidelines for percutaneous transluminal angioplasty. *Radiology* 1990;177:619-26.
 32. Palmaz JC. Intravascular stents: tissue-stent interactions and design considerations. *AJR Am J Roentgenol* 1993;160:613-8.
 33. Liu MW, Roubin GS, King SB III. Restenosis after coronary angioplasty: potential biologic determinants and role of intimal hyperplasia. *Circulation* 1989;79:1374-87.
 34. Ramsay LE, Waller PC. Blood pressure response to percutaneous transluminal angioplasty for renovascular hypertension: an overview of published series. *BMJ* 1990;300:569-72.
 35. Schreiber MJ, Pohl MA, Novick AC. The natural history of atherosclerotic and fibrous renal artery disease. *Urol Clin North Am* 1984;11:383-92.
 36. Strandness DE Jr. Natural history of renal artery stenosis. *Am J Kidney Dis* 1994;24:630-5.
 37. Novick AC, Ziegelbaum M, Vidt DG, Gifford RW Jr, Pohl MA, Goormastic M. Trends in surgical revascularization for renal artery disease: ten years' experience. *JAMA* 1987;257:498-501.
 38. Hallett JW Jr, Fowl R, O'Brien PC, et al. Renovascular operations in patients with chronic renal insufficiency: do the benefits justify the risks? *J Vasc Surg* 1987;5:622-7.
 39. Hansen KJ, Starr SM, Sands E, Burkart JM, Plonk GW Jr, Dean RH. Contemporary surgical management of renovascular disease. *J Vasc Surg* 1992;16:319-30.
 40. Weibull H, Bergqvist D, Bergentz SE, Jonsson K, Hulthen L, Manhem P. Percutaneous transluminal renal angioplasty versus surgical reconstruction of atherosclerotic renal artery stenosis: a prospective randomized study. *J Vasc Surg* 1993;18:841-52.

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