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Efficacy and safety of Palmaz stent insertion in the treatment of renal artery stenosis in kidney transplantation

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Abstract The aim of this study was to verify the safety and long-term efficacy of Palmaz stent insertion in the treatment of transplant renal artery stenosis (TRAS) in kidney transplantation. Nine of our transplanted patients were submitted to Palmaz stent insertion because of recurrence of renal artery stenosis after previous percutaneous transluminal angioplasty or because of severe ostial stenosis. The post-stenting results were excellent in all patients, with a follow-up period ranging from 1 to 3 years. The mean blood pressure (one-third systolic pressure plus two-thirds diastolic pressure) fell from 118.11 ± 7.44 to 103.21 ± 9.25 mm Hg; $P < 0.001$. Renal artery peak blood flow velocity as determined by Doppler sonography fell from 352 ± 73.24 cm/s to 169.8 ± 23.35 cm/s; $P < 0.001$. The serum creatinine 1-year after stenting was 1.3 ± 0.3 mg/dl with a slight reduction with respect to the pre-stenting values (1.5 ± 0.3 mg/dl; NS). As no complication occurred, we conclude that insertion of the Palmaz stent is a safe and effective way to treat recurrence of artery stenosis or ostial stenosis in renal transplanted patients.

Key words Palmaz stent · Renal artery stenosis · Renal transplantation · Vascular complication · Restenoses · Ostial stenoses

Introduction

Transplant renal artery stenosis (TRAS) is known to be the most frequent vascular complication in renal transplantation, with a reported incidence varying widely from 1% to 25% [7, 14, 31]. This wide range reflects both the skill of the surgical team and the heterogeneity of donor and recipient vessels and the methodology of detection of renal artery stenosis.

In relation to the arterial anastomotic site, the stenoses can be proximal, due in general to recipient atherosclerotic arterial disease, anastomotic or distal in the donor renal artery [25]. Anastomotic stenosis is usually regarded as of technical origin due to a faulty surgical technique and/or to postoperative fibrosis. The etiology of distal stenosis is less clear, but it may be related to mechanical or immunological damage [23].

Four different treatment modalities are currently available for patients with TRAS:

1. Medical management may be effective in controlling hypertension, but its effect on kidney function is not known. The conservative approach with medical management is generally indicated if the degree of stenosis is not hemodynamically significant and/or the degree of renal functional impairment is not significant [4]. On the other hand, there is agreement on the proposition that invasive correction of the stenosis is only justified when there is a reasonable expectation of success in improving hypertension and renal function or in stopping progressive renal deterioration [16, 17, 24, 30]. Because correction is not without danger to the graft, only severe stenoses are corrected in most centers. Roberts et al. suggest

that, in order to improve the results of correctional interventation, the physiological significance of a stenosis should be demonstrated prior to proceeding with an interventation that may present a risk to the transplanted kidney [24].

2. Surgical revascularization may be successful in treating TRAS, but this is a major operation with associated morbidity. Due to the technical difficulty of approaching vascular structures of an allograft that does not have collateral flow, the rate of graft loss after vascular reconstruction is nearly 30% [3, 13, 24]. Surgery has been indicated for a long period as the primary treatment in cases of kinking and proximal stenoses.
3. Percutaneous transluminal balloon angioplasty (PTA) has received considerable attention as a non-invasive treatment approach. This procedure achieves either a cure or an improvement in more than 70% of cases [11, 21]. Lower success rates have been reported and may be related to center experience or the type of lesion [8, 9, 24]. PTA is usually recommended as the procedure of choice for TRAS, that are short, linear and relatively distal from the anastomosis [3]. For ostial stenoses PTA has a lower rate of success [8] and carries a high risk of complication [28]. On the other hand, the recurrence rate of stenosis after PTA may be as high as 30% [3, 9–11, 21].
4. Insertion of metallic stents has recently been used to treat recurrent or ostial renal artery stenoses even if this procedure in the field of transplantation is still limited [18].

The aim of the study was prospectively to evaluate the efficacy and safety of stenting in the treatment of TRAS in nine cadaver kidney transplanted patients affected by arterial restenoses after previous PTA or severe ostial stenoses.

Patients and methods

We describe the Palmaz stent insertion and the biological, radiological and clinical follow-up in nine renal transplanted patients affected by restenoses or ostial stenoses. The patients were five males and four females, with a mean age of 47.3 ± 4 years, and transplant duration of 18.4 ± 18.6 months (range 2–48) before stenting. All patients had a severe TRAS documented by angiography. Four anastomoses were end-to-end and five anastomoses were end-to-side. The site of the stenoses was ostial in six patients. The follow-up period ranged from 17 to 47 months (Table 1).

Blood pressure, color Doppler sonography and serum creatinine levels were monitored before stenting and during the follow-up period for at least 1 year.

Statistical analysis of the data was performed with Student's *t*-test for quantitative variables. A value of $P < 0.05$ was considered statistically significant.

Table 1 Characteristics of patients and anastomoses. *A* anastomotic site stenosis, *P* post-anastomotic site stenosis, *E-S* end-to-side anastomosis, *E-E* end-to-end anastomosis

Patients	Gender	Age	Anastomosis	Site of stenosis	Time from Tx (months)	Follow-up (months)
1	M	55	E-E	A	48	35
2	M	40	E-E	A	2	33
3	F	48	E-E	P	6	22
4	F	30	E-E	A	6	32
5	M	54	E-S	A	28	36
6	M	42	E-S	A	48	46
7	M	49	E-S	A	18	47
8	F	49	E-S	P	3	30
9	F	59	E-S	P	7	17

Informed consent before interventation was obtained from all patients.

In all our patients, the lesion was dilated with a 4.8-French angioplasty balloon catheter passed through a valved 8-French introducer sheath with a femoral approach before placement of the stent. To evaluate the immediate technique result after angioplasty and to position the stent precisely, we introduced an aortic catheter through a contralateral femoral artery.

The intravascular stent was implanted over a stiff 0.5 mm guide wire that was left in the renal artery. In all patients, the Palmaz endoprosthesis was used. The stent was a stainless-steel tube which was crimped onto the same angioplasty balloon used for the previous angioplasty. Using a hockey-stick-shaped device, the guiding catheter-balloon-stent assembly was then passed over the guide wire across the lesion. The delivery system was then withdrawn into the aorta, leaving the stent in place mounted on the balloon. For accurate positioning of the endoprosthesis, angiography was performed with the catheter introduced contralaterally to adjust the position of the stent. In the case of ostial stenoses, the endoprosthesis was fitted to protrude 1–2 mm into the iliac or hypogastric arterial lumen. The balloon was then inflated and the stent was expanded to a diameter of 1–1.2 times that of the renal artery. The balloon was then removed and post-procedural angiography was performed.

Figures 1 and 2 show a pre-PTA and stent and a post-stent view of an ostial stenosis.

Before the procedure, 5000 IU of heparin was administered IV. Heparinization was maintained for 24 h at about 1250 IU per h, titrated to a level two to three times the normal partial thromboplastin time.

Hemostasis at the puncture site was achieved with manual compression. The patient remained in bed for 36 h. Ticlopidin (500 mg) was prescribed for 2 months and acetylsalicylic acid 300 mg without limit.

The technique is almost the same described by Blum et al. [5] for the treatment of ostial stenoses in the native renal artery.

Results

The mean follow-up period for the total study group was 33.5 ± 9.9 months (range, 17–47).

All stenting procedures were technically successful, with a reduction of trans-stenotic pressure gradient to less than 20 mm Hg.

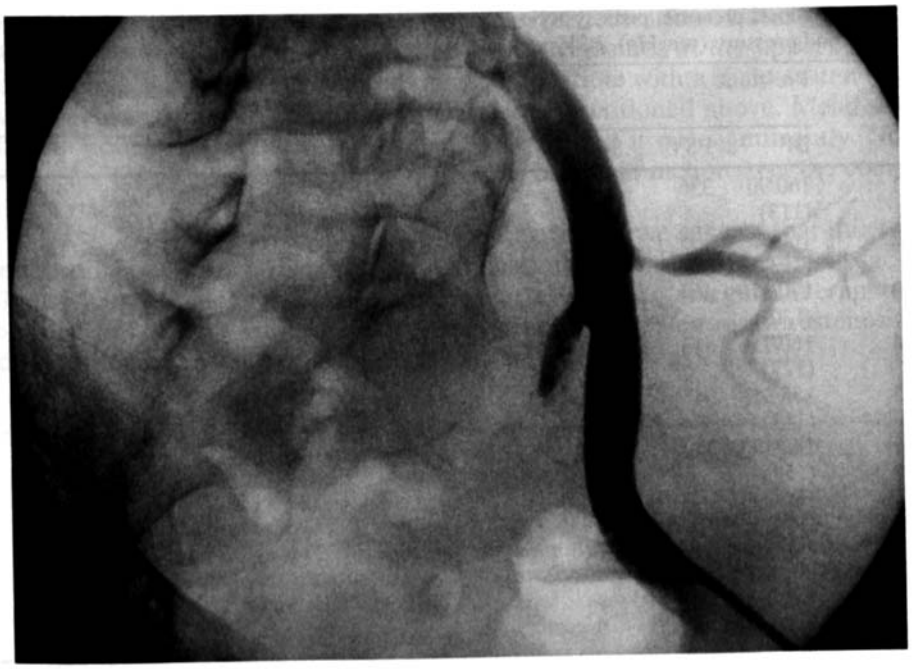

Fig. 1 Renal artery angiography: anastomotic stenosis pre-stenting

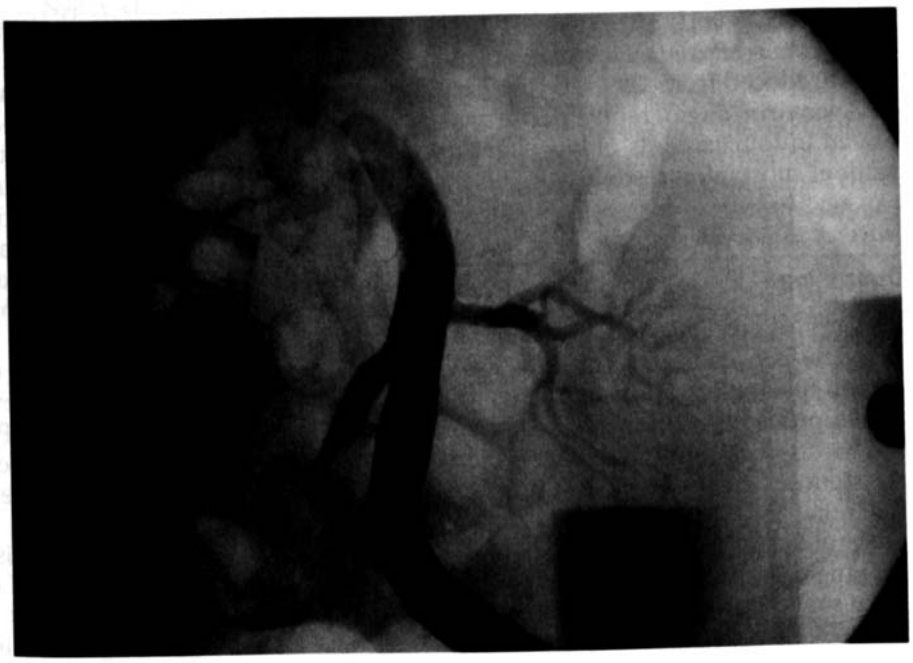

Fig. 2 Renal artery angiography: immediate post-stenting result of anastomotic stenosis

Significant procedure-related complications did not occur and no early or late major complications were verified.

Follow-up data on blood pressure, antihypertensive medication need, color duplex sonography and renal function were available for all patients and are shown in Table 2.

Patients showed a significant decrease of blood pressure with values of mean blood pressure of 118.7 ± 86.96 mm Hg pre stenting, 104.33 ± 14.87 after 1 week, 102.44 ± 8.72 after 1 month, 103.21 ± 9.25 after 1 year; $P < 0.001$. Antihypertensive medications required to maintain blood pressure under control decreased in seven patients out of nine. After 1 year from stenting, only two patients needed two antihypertensive medications.

The mean peak systolic velocity in the main renal artery as detected by color doppler sonography decreased from 363.44 ± 71.14 cm/s before stent insertion to

Table 2 Blood pressure, peak systolic velocity, serum creatinine and number of antihypertensive medications before and after stenting. *BP* blood pressure (mm Hg), *PSV* peak systolic velocity (cm/s), *SC* serum creatinine (mg/dl), *M* antihypertensive medications (number)

Pts	Pre-stent				1 week			Post-stent 1 month			1 year			
	BP	PSV	SC	M	BP	PSV	SC	BP	PSV	SC	BP	PSV	SC	M
1	160/90 (113)	336	2.1	1	140/80 (100)	197	1.9	130/80 (97)	205	1.8	140/80 (100)	180	1.8	0
2	160/95 (113)	505	2.1	2	120/80 (93)	143	1.8	140/90 (106)	160	1.5	120/80 (93)	175	1.4	1
3	170/110 (130)	464	1.3	2	140/80 (100)	156	1.2	140/70 (103)	170	1.2	170/100 (123)	190	1.2	1
4	160/110 (127)	313	1.1	3	150/100 (117)	170	1.1	150/100 (116)	190	1.2	140/100 (113)	190	1.2	2
5	155/95 (115)	345	1.5	3	150/95 (113)	135	1.4	150/95 (113)	140	1.4	150/80 (113)	140	1.4	1
6	150/100 (117)	340	1.5	1	140/70 (93)	190	1.3	140/70 (93)	210	1.3	150/85 (107)	190	1.1	1
7	160/100 (120)	348	1.6	2	120/80 (93)	100	1.7	130/90 (103)	160	1.4	170/105 (126)	162	1.5	2
8	155/85 (108)	300	0.8	2	120/70 (94)	160	0.8	120/70 (94)	160	0.7	130/80 (97)	177	0.9	1
9	190/90 (123)	320	1.8	1	170/120 (136)	58	2.2	150/85 (107)	85	2.6	130/80 (107)	125	2.0	1

145.44 ± 43.87 cm/s after 1 week, to 169.8 ± 23.35 cm/s after 1 year; $P < 0.001$. Mean serum creatinine after 1 year reduced from 1.5 ± 0.3 to 1.3 ± 0.3 mg/dl; NS.

A control angiography performed after 1 year revealed in four patients only a slight intima hyperplasia without substantial restenosis.

Discussion

As surgical correction of TRAS has a graft loss rate of about 30% even in expert hands, PTA is now a well accepted revascularization technique [6]. PTA is a less invasive procedure, can be repeated early in the case of restenosis and represents the preferred option when the stenoses are in the hilar or distal portion of the arterial renal bed. However, even if many reports in the literature address the role of percutaneous transluminal angioplasty in patients with TRAS and some of them suggest that angioplasty should be the initial treatment of choice for all such patients [27, 29], this technique is not free of complications and failure that have been reported to occur more frequently than with surgery [24].

Most serious procedure-related complications of PTA are major injury to the renal artery, resulting in thrombosis or intimal flaps. For stenoses at the anastomotic line, PTA has a lower rate of success [8] and carries a high risk of complications [28]. Another serious limitation to PTA is the elastic recoil of the arterial wall, which leads to restenosis with rates up to 33% [1].

To overcome the problem of elastic recoil after angioplasty, recent reports recommend different types of intravascular stents for the treatment of non-ostial renal artery stenoses both in native and in transplanted kidneys [12, 15]. Metallic stents have been used to treat ostial stenoses in native kidneys [5, 22] and more recently in transplanted kidneys as well [18, 23]. In some cases, stent placement has been suggested to provide effective non-operative management of flow compromising dissection induced by PTA [2] and of vessel reclosure during PTA.

We have now described nine renal transplanted patients who had severe renal artery restenoses after previous successful PTA or severe ostial stenoses. All patients were treated with the Palmaz metallic stent insertion and were prospectively followed for at least 1 year.

Use of stents is a generally recognized procedure in the treatment of ostial lesions in native kidneys or in the management of complications after renal artery PTA (occlusive dissections, early elastic recoil). The relevant literature describes use of stents also in kidney graft artery dilatation [19, 26].

We were aware of complications due to stent implantation. Acute complications most frequently reported are thrombosis, groin hematomas, peripheral embolization, transient renal failure, subintimal dissection, stent misplacement or mobilization. Late complications are mostly represented by restenosis, usually caused by myointimal hyperplasia. 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 [20].

In our nine patients we had no acute complications and prompt correction of the stenoses was achieved, as documented by the control angiography, the normalization of mean peak systolic blood flow at color Doppler sonography and optimal results on the hypertensive status.

All patients were carefully monitored throughout the follow-up period ranging from 17 to 47 months. Clinical and radiological data at 1 year were optimal for all patients, with a stable reduction of blood pressure and reduction of antihypertensive medications. Color Doppler sonography showed peak velocities at the upper limits of the normal range, compatible with a slight restenosis due to the healing process mentioned above. Mean serum creatinine improved, even if not significantly. No patient had a worsening of renal function over the complete follow-up period.

According to our experience, we suggest that due to its acceptable complication rate and high technical success, transplant renal artery stenting is a good therapeutic approach to treatment of ostial stenosis or restenosis after previous PTA.

References

1. Baert AL (1994) Renal artery stent placement. Radiology 191: 619–621
2. Becker GJ (1991) Intravascular stents: general principles and status of lower extremity arterial applications. Circulation 83: I-122–I-136
3. Benoit G, Moukarzel M, Hiesse C, Verdelli G, Charpentier B, Fries D (1990) Transplant renal artery stenosis: experience and comparative results between surgery and angioplasty. Transplant Int 3: 137–140
4. Beyga ZT, Kahan BD (1998) Surgical complications of kidney transplantation. J Nephrol 11: 137–145
5. Blum U, Krumme B, Flugel P, Gabelmann A, Lehnert T, Buitrago-Tellez C, Schollmeyer P, Langer M (1997) Treatment of ostial renal artery stenoses with vascular endoprostheses after unsuccessful balloon angioplasty. N Engl J Med 336: 459–465
6. Boisclair C, Therasse E, Oliva VL, Soulez G, Bui BT, Querin S, Robillard P (1997) Treatment of renal angioplasty failure by percutaneous renal artery stenting with Palmaz stent: midterm technical and clinical results. Am J Rontgenol 168: 245–251
7. Chan HWH, Ho YW, Chan CM, Yiu TF, Tong MKL, Wong PHC (1995) Treatment of anastomotic ostial allograft and renal artery stenosis with the Palmaz stent. Transplantation 59: 439
8. Chandrasoma P, Aberle AM (1986) Anastomotic line renal artery stenosis after transplantation. J Urol 135: 1159–1162
9. Clements R, Evans C, Salaman JR (1987) Percutaneous transluminal angioplasty of renal transplant artery stenosis. Clin Radiol 38: 235–237
10. Fauchald P, Vatne K, Paulsen D, Brodahl U, Sodal G, Holdaas H, Berg KJ, Flatmark A (1992) Long term clinical results of percutaneous transluminal angioplasty in transplant renal artery stenosis. Nephrol Dial Transplant 7: 256–259
11. Greenstein S, Verstanding A, Mc Lean G, Dafoe DC, Burke DR, Meranze SG, Naji A, Grossman RA, Perloff LJ, Barker CF (1987) Percutaneous transluminal angioplasty.Transplantation 43: 29–32
12. Henry M, Amor M, Henry I (1996) Stent placement in the renal artery: three-year experience with the Palmaz stent. J Vasc Interv Radiol 7: 343–350
13. Hohnke C, Abendroth D, Schleibner S, Land W (1987) Vascular complications in 1200 kidney transplantations. Transplant Proc 19: 3691–3692
14. Loubeyre P, Cahen R, Grozel F, Trolliet P, Pouteil-Noble C, Labeeuw M (1996) Transplant renal artery stenosis. Transplantation 62: 446–450
15. Mac Leod M, Taylor AD, Baxter G (1995) Renal artery stenosis managed by Palmaz stent : technical and clinical outcome. J Hypertens 13: 1791–1795
16. Merkus JWS, Huysmans FTM, Hoitsma AJ, Buskens FGM, Skotnicki SH, Koene RAP (1993) Renal allograft artery stenosis: results of medical treatment and intervention. A retrospective analysis. Transplant Int 6: 111–115
17. Merkus JW, Hoitsma AJ, van Asten JC, Zeebregts AM, van der Vliet JA, Strijk SP, Koene RA, Skotnicki SH (1995) Echo-Doppler diagnosis of renal allograft artery stenosis. Clin Transplant 9: 383–389
18. Newman-Sanders APG, Gedroyc WG, Al-Kutouby MA, Koo C, Taube D (1995) The use of expandable metal stents in transplant renal artery stenosis. Clin Radiol 50: 245–250
19. Peregrin JP, Lacha Jiri, Adamec M (1998) Successful handling by stent implantation of postoperative renal graft artery stenosis and dissection. Nephrol Dial Transplant 13: 1004–1006
20. Palmaz JC (1993) Intravascular stent: tissue-stent interactions and design considerations. Am J Rontgenol 160: 613–618
21. Raynaud A, Bedrossian J, Remy P, Brisser JM, Angel CY, Gaux JC (1986) Percutaneous transluminal angioplasty of renal transplant arterial stenosis.Am J Roentgenol 146: 853–857
22. Rees CR, Palmaz JC, Becker GJ, Ehrman KO, Richter GM, Noeldge G, Katzen BT, Dake MD, Scwarten DE (1991) Palmaz stent in atherosclerotic stenoses involving the ostia of the renal arteries: preliminary report of a multicenter study. Radiology 181: 507–514
23. Rengel M, Da Silva GG, Inchaustegui L, Lampreave JL, Robledo R, Echenagusia A, Vallejo JL,Valderrabano F (1998) Renal artery stenosis after kidney transplantation: diagnostic and therapeutic approach. Kidney Int 54: S99–S106
24. Roberts JP, Asher NL, Fryd DS, Hunter DW, Dunn DL, Payne WD, Sutherland DER, Dastanega-Zuniga W, Najarian JS (1989) Transplant renal artery stenosis. Transplantation 48: 580–583
25. Sankari BR, Geisinger M, Zelch M, Brouhard B, Cunningham R, Novick AC (1996) Post-transplant renal artery stenosis: impact of therapy on long term kidney function and blood pressure control. J Urol 155: 1860–1864
26. Savio D, Rossato D, Margarita D, Messina M, Rabbia C (1996) Renal transplantation: evaluation of stenting in the treatment of restenosis and failure of previous transluminal angioplasty. Cardiovasc Intervent Radiol 19: S77

27. Sos TA, Pickering TG, Sniderman K, Saddekni S, Case DB, Silane MF, Vaughan ED Jr, Laragh JH (1983) Percutaneous transluminal renal angioplasty in renovascular hypertension due to atheroma or fibromuscolar dysplasia. N Engl J Med 309: 274–279
28. Sutherland RS, Spees IK, Jones JW, Fink DW (1993) Renal artery stenosis after renal transplantation: the impact of the hypogastric artery anastomosis. J Urol 149: 980–985
29. Tegtmeyer CJ, Kellun CD, Ayers C (1984) Percutaneous transluminal angioplasty of the renal artery: results and long-term follow-up. Radiology 153: 77–84
30. Tilney NL, Rocha A, Strom TB, Kirkman RL (1984) Renal artery stenosis in transplant patients. Ann Surg 199: 454–460
31. Wong W, Fynn SP, Higgins RM, Walters H, Evans S, Deane C, Gross D, Bewick M, Snowden SA, Scoble JE, Hendry BM (1996) Transplant renal artery stenosis in 77 patients. Does it have an immunological cause? Transplantation 61: 215–219