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Restenosis Rates After Drug-Eluting Stent Treatment for Stenotic Small-Diameter Renal Arteries

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Abstract
Purpose To determine primary rates in small-diameter renal arteries, including complex bifurcation lesions, treated with drug-eluting stents (DES) in patients with atherosclerotic renal artery stenosis.
Materials and Methods This is a retrospective single-institution study. A total of 37 patients with 39 stented renal arteries were included. Patient and procedural data were obtained from the electronic medical record. Survival free from restenosis was estimated using the Kaplan–Meier method with patients stratified into two groups based on renal artery diameters ($\leq$ 3.5 mm or $>$ 3.5 mm). Univariate Cox proportional models were used to estimate hazard ratios associated with clinical and angiographic variables.
Results Average renal artery diameter at time of treatment was 3.4 mm $\pm$ 0.4 mm. The median survival free from restenosis was 992 days, with 11 out of 37 (29.7%) developing an in-stent restenosis. Renal arteries $<$ 3.5 mm in diameter had similar patency rates as renal arteries $>$ 3.5 mm (P = 0.33). The 1-, 2-, and 5-year patency rates were 71%, 63%, and 38%, respectively. History of stroke was the only comorbidity to portend a significantly greater rate of restenosis (hazard ratio 3.77; 95%CI, 1.05–13.6; P = 0.04). Medications did not statistically alter the risk of restenosis.
Conclusion Revascularization of renal arteries with DES achieved similar primary patency rates irrespective of renal artery diameter. Stent configuration was not associated with time to renal replacement therapy or all-cause mortality.
Level of Evidence Level 3, Cohort Study.

Keywords Drug-eluting stent · Renal artery stenosis · Renal replacement therapy

Introduction

Atherosclerotic renovascular disease (ARVD) accounts for 90% of all cases of renal artery stenosis (RAS) [1]. The estimated prevalence of clinically silent ARVD in healthy individuals 65 years and older is 7% [2]. As expected, the prevalence of ARVD is greater (approximately 12–50%) in subsets with hypertension, documented systemic atherosclerosis, and/or cardiovascular risk factors [3].

If left untreated, ARVD and RAS can lead to renal insufficiency, medically refractory hypertension, and circulatory congestion [2]. The current American Heart Association (AHA) guidelines have established several class IIa indications for renal artery revascularization in the management of blood pressure control and renal function preservation. For blood pressure control, these indications include: accelerated or malignant hypertension, hypertension with medication intolerance, and hypertension in the

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setting of unexplained unilateral renal atrophy [4]. For renal function preservation, these indications include progressive chronic kidney disease (CKD) with bilateral RAS and a solitary kidney with RAS. Per the AHA guidelines, revascularization may be considered in patients with CKD and unilateral ARVD, though this is a class IIb indication [4].

Bare metal stents (BMS) are the most commonly used scaffold for endovascular renal artery revascularization, with restenosis rates around 15% at 6 months. Similar to coronary arteries, the most reliable predictor of restenosis with BMS in renal arteries is small artery or stent diameter [5]. At present, there are limited data suggesting drug-eluting stents (DES) may have better patency when placed in the renal artery for ARVD [6]. The impact of drug-eluting technology may be particularly beneficial in smaller caliber arteries in which a given volume of neointimal hyperplasia obstructs a greater luminal cross-sectional area compared to larger arteries. In this single-institution retrospective study, we aim to determine in-stent restenosis rates among DES deployed for ARVD in small-diameter renal arteries ($\leq$ 4 mm). Additionally, we examined the risk factors associated with in-stent restenosis, time to renal replacement therapy (RRT), and all-cause mortality.

Materials and Methods

Institutional review board approval was obtained prior to the initiation of this retrospective study. All patients who underwent DES placement for the treatment of ARAS from June 2004 to May 2017 were reviewed. Patient demographics, comorbidities, use of certain medications, type and size of DES placed, stent configuration, restenosis rates, and length of follow-up were obtained from the electronic medical record. Fifty-nine patients were treated with DES for renal artery stenosis. Patients treated for renal allograft artery stenosis (n = 3) or in-stent restenosis (n = 15) were excluded. Four additional patients denied research authorization and were excluded. After application of exclusion criteria, the study group included 37 patients and 39 renal arteries.

The primary aim of this study was to identify the time to restenosis. The secondary aims were to determine rates of progression to renal replacement therapy and all-cause mortality. After DES placement, follow-up was recommended at 3-, 6-, and 12-month intervals and yearly thereafter. These follow-up appointments occur with the performing interventional radiologist and include blood pressure and renal function assessment as well as a renal ultrasound with Doppler. Data regarding renal replacement therapy (hemodialysis, peritoneal dialysis, or renal transplantation) were directly obtained from these follow-up appointments. Data on all-cause mortality were obtained either directly from follow-up appointments, or in those lost to follow-up (7/37 or 19%), the United States Social Security Death Index (SSDI).

Definitions

Glomerular filtration rate (GFR) was estimated using the serum creatinine level obtained before the renal artery stent procedure using the Modification of Diet in Renal Disease formula [7]. Baseline creatinine data were collected and analyzed based on the most recent creatinine obtained within 30 days before the stent procedure. Each patient's CKD stage was based on his or her GFR at the time of first DES placement. The CKD stage was determined based on the Kidney Disease Improving Global Outcomes (KDIGO) CKD Work Group classification [8].

Renal artery stenosis was defined as a velocity $\geq$ 200 cm/s on duplex ultrasonography and/or $>$ 50% stenosis estimated visually at time of renal catheter angiography. In-stent renal artery stenosis was defined as a velocity $\geq$ 250 cm/s on duplex ultrasonography and/or $>$ 50% stenosis estimated visually at time of renal catheter angiography [9, 10].

Drug-Eluting Stent Placement

The decision to use a DES, rather than a bare metal stent, was left to the interventionalist's discretion. The interventional procedure has been described in detail elsewhere, including the technique utilized for the placement of kissing stents when treating complex renal artery bifurcation lesions [11]. A total of 39 renal arteries were treated with DES [Cypher (Cordis, Milpitas, California), n = 17; Taxus (Boston Scientific, Marlborough, Massachusetts), n = 9; Xience (Abbott, Abbott Park, Illinois), n = 8; Promus (Boston Scientific), n = 3; Synergy (Boston Scientific), n = 1; Resolute, n = 1 (Medtronic, Minneapolis, Minnesota)] for renal artery stenosis. All stents were placed in arteries with diameters 4 mm or smaller. Only 2 out of 37 patients had bilateral stents placed. Technical success was defined as stent deployment with full expansion to the nominal stent diameter.

Statistical Methods

Descriptive statistics for categorical variables are expressed as numbers and percentages. Continuous variables are expressed as mean $\pm$ SD or medians and ranges as appropriate. Survival was estimated using the Kaplan–Meier method for the primary outcome: survival free from restenosis, and secondary outcomes: all-cause mortality and survival free from RRT. Univariate Cox proportional

hazards models were used to estimate the hazard ratios (HRs) associated with clinical and angiographic variables before the intervention including CKD stage, diabetes mellitus, hyperlipidemia, stroke, coronary and carotid artery disease, peripheral artery disease, smoking status, and use of certain medications including statins, antiplatelet agents, angiotensin-converting enzyme inhibitors (ACEIs), and angiotensin receptor blockers (ARBs). Given the limited number of events for each of the outcomes, we did not examine any multiple variable models. All analyses were completed using SAS version 9.4 software (SAS Institute Inc., Cary, North Carolina).

Results

Twenty-two (59.5%) patients were female, with a total mean age of 73.4 years ± 9.1 years (range 46–89 years). The median follow-up period was 1.3 years (range 0–12.4 years). Patient demographics are summarized in Table 1. Technical success was achieved in for all stents deployed (100%).

Time to Restenosis

The median survival free from restenosis was 992 days, with 11/37 (29.7%) developing an in-stent restenosis. By Kaplan–Meier estimates, the 1-, 2-, and 5-year patency rates were 71%, 63%, and 38%, respectively. Patency rates were similar at 1, 2, and 5 year for patients with renal artery diameters ≤ 3.5 mm compared to diameters > 3.5 mm (Fig. 1; $P = 0.33$).

Table 1 Baseline patient characteristics

Variable	Value
Age	73.4 ± 9.1
Female sex (%)	59.5%
CKD stage	
0/1/2/3A	27.1%
3B/4/5	72.9%
Proteinuria (mg/24 h)	573.5 ± 1150.8
Hypertension	97.3%
Diabetes mellitus	35.1%
Hyperlipidemia	75.7%
History of stroke	27%
Coronary artery disease	62.2%
Peripheral artery disease	29.7%
Smoking status	38.2%
Current or former	
ACEI/ARB	45.9%
Antiplatelet	91.9%
Statin	81.1%

Thirty-nine renal arteries stenoses were treated, and 14 (36%) of which were main renal artery ostial lesions. Stents were placed in six (15%) accessory renal arteries and 15 (38%) segmental renal arteries. Five of the 15 segmental arteries (33%) were treated with a "kissing stent" configuration. The four (10.3%) remaining renal arteries had stents placed for non-ostial main renal artery stenosis. In-stent restenosis risk did not differ based on stent location or configuration. None of the patients treated with kissing stents for complex bifurcation lesions developed a restenosis.

Univariate analysis was used to identify risk factors that may be associated with restenosis (Table 2). History of stroke was the only comorbidity to portend a significantly greater risk of restenosis (hazard ratio 3.77; 95%CI, 1.05–13.6; $P = 0.04$). Though not significant, the absolute number of restenoses was greater in patients with hyperlipidemia, coronary artery disease, peripheral artery disease, and CKD stage 3 or higher. While a majority of the patients were on statins, antiplatelet agents, and ACEI/ARBs, none of these were significantly associated with an increased risk of restenosis.

Time to Renal Replacement Therapy

Four patients ultimately progressed to RRT (hemodialysis, $n = 3$; renal transplantation, $n = 1$) after DES placement. The number of males and females progressing to RRT was equal (males, $n = 2$; females, $n = 2$). In univariate analysis, high-grade proteinuria (HR = 1.001; 95%CI, 1.000–1.001; $P = 0.022$) and high baseline creatinine (HR 14.6; 95%CI, 1.691–125.390; $P = 0.015$) were associated with progression to RRT. Other factors such as diabetes, hyperlipidemia, smoking status, coronary, carotid and peripheral artery disease, stroke, and use of statins, antiplatelet agents, and ACEI/ARBs were not significantly associated with progression to RRT. All univariate results are listed in Table 3.

All-Cause Mortality

There were 16 patient deaths in the study group. The median survival after stent placement was 6.7 years (range 0–12.6 years). In univariate analysis, using CKD stage 1/2 as the reference group, CKD stage 4/5 (HR = 10.69; 95%CI, 1.29–88.57; $P = 0.028$) was associated with increased risk of all-cause mortality. CKD stage 3A/3B did not significantly increase the risk of all-cause mortality. Other factors included stroke (HR = 3.150; 95%CI, 1.082–9.174; $P = 0.035$) and higher baseline creatinine (HR = 2.381; 95%CI, 1.324–4.282; $P = 0.004$). Additional

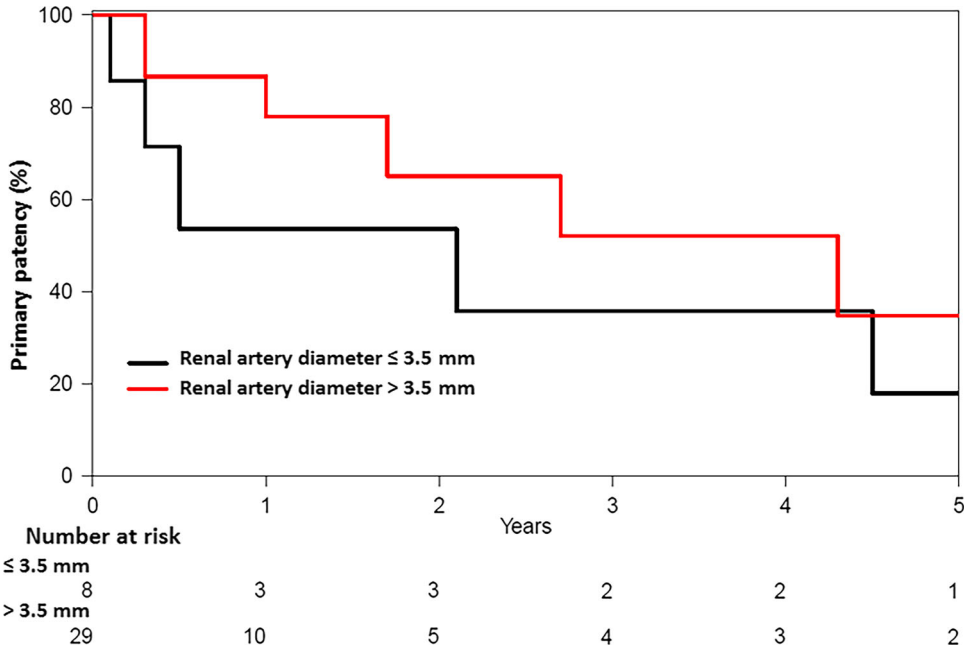

Fig. 1 Effect of renal artery diameter on primary patency after DES placement. Primary patency rates were less at nearly every time point for renal artery diameters ≤ 3.5 mm ($P = 0.33$)

comorbidities and medications were not significantly associated with an increased risk of all-cause mortality. Stent configuration was not associated with a difference in all-cause mortality. These analyses are summarized in Table 4.

Complications

The complication rate was 5.4% (2/37 patients). This included one case of in situ thrombosis of the distal main renal artery after stent placement and another case of distal segmental renal artery embolization. Both of these were treated with intra-arterial thrombolysis with tissue plasminogen activator (tPA). Immediate follow-up angiography showed near complete resolution of thrombus in each case. The 30-day all-cause mortality rate was 2.7% (1/37 patients). The one death was related to multi-organ failure in the setting of significant left ventricular outflow tract obstruction. The death was not related to the renal artery stent procedure.

Discussion

This cohort of patients with ARVD and small-diameter renal arteries (≤ 4 mm) treated with DES represents the largest to date [10]. No significant risk factor for in-stent restenosis was identified, which may be due to the limited number of restenosis events ($n = 11$). Proteinuria and poor renal function at the time of intervention had significant association with the time to RRT. Finally, advanced CKD and history of stroke increased the risk of all-cause mortality for patients with stenotic small-diameter renal arteries treated with DES.

Drug-eluting stents have been traditionally used for coronary artery stenosis and are associated with lower restenosis rates [10]. The DES are composed of a metallic stent platform, a polymer coating, and an antiproliferative agent. The polymer coating serves to both carry and control the release of the antiproliferative agent. First-generation paclitaxel-eluting stents (Taxus, Boston Scientific) and sirolimus-eluting stents (Cypher, Cordis) achieved a significant reduction in risk of restenosis and target lesion revascularization (TLR) over BMS, but were prone to very late stage (> 1 year) thrombosis. Second-generation everolimus-eluting stents (Promus, Synergy, and Xience, Boston Scientific) and zotarolimus-eluting stents (Synergy, Medtronic) have improved the safety profile of DES through the use of novel stent materials, platforms, and delivery systems while maintaining the efficacy of first-generation DES [12, 13].

In the GREAT trial, renal artery in-stent restenosis rates after the use of 5–6 mm drug-eluting or bare metal stents were compared. Drug-eluting stents were associated with a 50% relative risk reduction in in-stent restenosis when compared to BMS [14]. These results were further demonstrated in two additional retrospective studies. Our group previously performed a preliminary retrospective study comparing restenosis rates in renal arteries 4 mm or smaller after treatment with either DES or BMS. The patency rates at 1 and 2 years for DES were 78% and 68%, respectively, versus 58% and 47% for BMS. While DES demonstrated improved primary patency, the results did not reach significance, likely due to small patient numbers (18

Table 2 Cox proportional hazard models—survival free from restenosis

Risk factors	No. of patients with data		No. of restenosis	Univariable	
				HR (95% CI)	*P* value
Sex (male)	Male	15	4	0.82 (0.24–2.84)	0.758
	Female	22	7		
CKD stage 3A/3B	17		9	1.95 (0.25–15.54)	0.527
4/5	14		1	1.70 (0.10–29.33)	0.713
0/2	6		1	1.0 (reference)	
Diabetes mellitus	Yes	13	4	0.81 (0.23–2.82)	0.740
	No	24	7		
Hyperlipidemia	Yes	28	8	1.42 (0.37–5.39)	0.609
	No	9	3		
Stroke	Yes	27	7	3.77 (1.05–13.61)	0.043
	No	10	4		
Coronary artery disease	Yes	23	9	3.969 (0.81–19.34)	0.088
	No	14	2		
Carotid artery disease	Yes	10	3	1.79 (0.47–6.91)	0.397
	No	27	8		
Smoking	Former	13	4	1.25 (0.35–4.48)	0.735
	Never	21	6		
Peripheral artery disease	Yes	11	3	1.00 (0.26–3.84)	0.996
	No	26	8		
Statin	Yes	30	8	0.73 (0.19–2.77)	0.641
	No	7	3		
Antiplatelet	Yes	34	10	1.72 (0.22–13.60)	0.606
	No	3	1		
ACEI/ARB	Yes	17	6	0.94 (0.28–3.16)	0.918
	No	20	5		
Stent configuration					
Main renal artery	18		6	0.81 (0.25–2.69)	0.733
Segmental renal artery	19		5	1.0 (reference)	

CKD chronic kidney disease; *ACEI/ARB* angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; *HR* hazard ratio; *CI* confidence interval

patients in the BMS group and nine in the DES group) [10]. A larger retrospective study by Baradaric et al. compared restenosis rates in a comparable subset of patients with maximum renal artery balloon sizes of 4–6.5 mm in diameter after DES, BMS-in-DES hybrid technique, or BMS placement. They found a statistically significant reduction in restenosis rates at 1 year for lesions treated with DES as compared to BMS (7.2 vs 18.6%), with no restenosis in the lesions treated with the hybrid technique [15].

We observed primary patency rates of 71% and 63% at 1 and 2 years, respectively, in 39 renal arteries with an average renal artery diameter of 3.4 mm ± 0.4 mm. Risk of restenosis did not differ based on stent location or configuration. All five complex bifurcation lesions treated with a "kissing DES technique" were free from restenosis. These primary patency rates are slightly lower than those demonstrated in studies by Misra et al. [10] and Baradaric et al. [15]. While average renal artery diameters were identical between the current study and Misra et al. (3.4 mm ± 0.4 mm vs 3.4 mm ± 0.4 mm), only 4–6.5 mm balloon sizes were included in Baradaric's study [15]. Of note, DES in our study achieved better primary patency rates when compared to the BMS primary patency rates in those studies, even when accounting for renal artery size differences.

One of the known risks of restenosis is small renal artery diameters [14]. A retrospective study by Lederman et al. examined restenosis rates after bare metal stenting of the renal arteries in 363 patients at a median follow-up of 16 months. While no baseline clinical markers predicted restenosis, there was a significant increase in restenosis rates with decreasing renal artery diameter. The restenosis rate was only 6.5% with artery diameters > 6 mm, but

Table 3 Cox proportional hazard models—survival free from RRT

Risk factors	No. of patients with data		No. of RRT	Univariable	
				HR (95% CI)	*P* value
Sex	Male	15	2	1.24 (0.17–8.86)	0.830
	Female	22	2		
CKD stage 3A/3B	17		1	0.75 (0.02–31.58)	0.882
4/5	14		3	20.54 (0.30–1387.70)	0.160
0/2	6		0	1.0 (reference)	
Diabetes mellitus	Yes	13	1	0.52 (0.05–5.09)	0.576
	No	24	3		
Hyperlipidemia	Yes	28	3	1.30 (0.14–12.49)	0.821
	No	9	1		
Stroke	Yes	10	1	1.38 (0.14–13.27)	0.782
	No	27	3		
Coronary artery disease	Yes	23	1	0.20 (0.02–1.91)	0.161
	No	14	3		
Carotid artery disease	Yes	10	1	1.05 (0.11–10.25)	0.965
	No	27	3		
Smoking	Former	13	2	3.18 (0.28–35.72)	0.348
	Never	21	1		
Peripheral artery disease	Yes	11	1	0.79 (0.08–7.64)	0.838
	No	26	3		
Statin	Yes	30	4	2.63 (0.10–71.02)	0.565
	No	7	0		
Antiplatelet	Yes	34	4	1.03 (0.04–28.03)	0.984
	No	3	0		
Stent configuration	37				
Main renal artery	18		2	0.99 (0.14–7.15)	0.995
Segmental renal artery	19		2	1.0 (reference)	

CKD chronic kidney disease; *HR* hazard ratio; *CI* confidence interval

36% for artery diameters < 4.5 mm [16]. In the coronary arteries, DES have shown consistently improved primary patency rates when compared to BMS, even in arteries < 3 mm in diameter [17]. When considering the data presented by Lederman et al. [16], the overall restenosis rate of 29.7% in our study is encouraging given the very small diameters of these treated lesions.

While the ASTRAL, STAR, and CORAL trials all failed to demonstrate a benefit for renal artery revascularization over intensive, multitargeted vascular protection therapy alone, none of these studies looked at restenosis rates, an important factor in considering the mechanistic success of the intervention [18, 19]. Future prospective studies should consider the type and configuration of the stent used, the arterial diameters of treated lesions, and restenosis rates for each patient subset before appraising the efficacy of endovascular renal artery interventions.

In our study, higher baseline creatinine increased both the risk of death and risk of progression to RRT. High-grade proteinuria, unlike higher baseline creatinine, only increased the risk of progression to RRT. High-grade proteinuria and higher baseline creatinine did not portend a greater risk of restenosis. Elevated creatinine and high-grade proteinuria are both markers of renal impairment and have been shown to be associated with increased risk of mortality and progression to RRT in prior studies [20–23].

Patients with a history of stroke had higher rates of in-stent restenosis and mortality. This is a logical finding given that stroke is generally associated with vascular disease and a major cause of cardiovascular mortality. In contrast, stroke was not a significant risk factor for mortality in a study involving over 1000 patients who had received bare metal renal artery stents [20]. Cerebrovascular disease history was a significant risk factor for cardiovascular mortality but not all-cause mortality in a study by Wallace et al. [24]. Renal artery stenosis is a common manifestation of atherosclerosis and is frequently associated with coronary, cerebrovascular, and peripheral

Table 4 Cox proportional hazard models—all-cause mortality

Risk factors	No. of patients with data		No. of deaths	Univariable	
				HR (95%CI)	*P* value
Sex	Male	15	6	0.72 (0.26–1.98)	0.518
	Female	22	10		
CKD stage 3A/3B	17		6	1.06 (0.12–9.48)	0.961
4/5	14		9	10.69 (1.29–88.57)	0.028
0/2	6		1	1.0 (reference)	
Diabetes mellitus	Yes	13	7	0.88 (0.33–2.39)	0.806
	No	24	9		
Hyperlipidemia	Yes	28	13	1.30 (0.36–4.70)	0.687
	No	9	3		
Stroke	Yes	10	8	3.15 (1.08–9.17)	0.035
	No	27	8		
Coronary artery disease	Yes	23	13	1.54 (0.43–5.56)	0.507
	No	14	3		
Carotid artery disease	Yes	10	4	1.86 (0.27–2.71)	0.801
	No	27	12		
Smoking	Former	13	6	1.00 (0.32–3.14)	0.998
	Never	21	8		
Peripheral artery disease	Yes	11	5	1.00 (0.26–3.84)	0.921
	No	26	11		
Statin	Yes	30	13	0.79 (0.22–2.85)	0.715
	No	7	3		
Antiplatelet	Yes	34	15	0.68 (0.09–5.38)	0.714
	No	3	1		
Stent configuration	37				
Main renal artery	18		6	1.38 (0.50–3.85)	0.537
Segmental renal artery	19		10	1.0 (reference)	

CKD chronic kidney disease; *HR* hazard ratio; *CI* confidence interval

vascular diseases. History of stroke in our patient population likely serves as a marker of significant comorbid cardiovascular disease and therefore is a significant risk factor for all-cause mortality [25].

Chronic kidney disease stage was not a significant risk factor for restenosis risk or progression to renal replacement therapy. In our study, CKD stage 4 or higher increased the risk of all-cause mortality. These data are similar to a previous study of ours, though patients in that study with CKD stage 3B were also at an increased risk of death from all causes [20].

Stent location and configuration did not portend a greater risk of restenosis, all-cause mortality, or progression to RRT; however, adequate power in this study was not achieved to detect a difference between the patient subsets. Larger trials in the future should stratify patients based on stent location and configuration to determine whether these factors predict patient outcomes; this information would be very valuable for procedure planning.

This study has several limitations, many of which are inherent to retrospective studies including selection and recall bias, lack of defined follow-up period with patient loss to follow-up, variability in pre- and post-intervention laboratory and clinical data collection, and lack of procedure standardization. The number of patients and restenosis events in this study was relatively small. This results in fairly wide confidence intervals and limited data conclusions, which would be remedied by larger studies.

Conclusion

In conclusion, patients with small-diameter renal arteries represent a complex subset of patients with ARVD and RAS and are at great risk of restenosis. Elevated baseline creatinine is an important risk factor for subsequent mortality and progression to RRT after renal artery stent placement. History of stroke serves as a marker of

significant cardiovascular disease and portends a greater risk of restenosis and mortality.

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Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent This study has obtained IRB approval from the Mayo Clinic IRB committee, and the need for informed consent was waived.

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