EDITORIAL

Does stenting for atherosclerotic renovascular disease improve blood pressure and kidney function better than medical treatment?

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Atherosclerotic renovascular disease (ARD) is a common condition in which the atherosclerotic narrowing of renal arteries may lead to renin-dependent hypertension, progressive renal dysfunction, and/or recurrent pulmonary edema.¹ It typically occurs in high-risk patients with coexistent vascular disease elsewhere.² Consequently, most patients with ARD are likely to die from coronary heart disease or stroke before end-stage renal failure occurs. The incidence of renal artery thrombosis is less than 1% per year.^{3,4} The risk of chronic renal replacement therapy is 18 times lower than that of a major cardiovascular event.⁵⁻⁷

Stenting aims at reducing blood pressure (BP), stabilizing or improving renal function, and preventing cardiovascular and renal events in patients with ARD. Controlled trials comparing medication plus stenting to medication alone, medication plus surgery, or medication plus angioplasty without stenting provided disappointing results (TABLE).⁸⁻¹¹ Most patients undergoing stenting to treat hypertension associated with ARD, still require antihypertensive agents after the procedure because the reduction in BP following stenting is modest, and because several antihypertensive agents, such as renin-angiotensin antagonists or β -blockers, are needed to prevent cardiovascular and renal events even in patients with normalized BP. Two recent trials compared renal outcome in ARD patients provided with medication plus stenting with that in ARD patients supplied with medication alone.^{11,12} Their results have shown that stenting does not preserve renal function. Improvements in revascularization techniques did not alter BP or renal outcomes of angioplasty. Compared with angioplasty alone, angioplasty plus stenting improved renal artery patency but did not improve BP control or renal function.⁸ Compared with stenting

alone, stenting plus protection devices and intravenous platelet inhibition did not improve renal function.⁹ The main explanation for these negative results is that ARD involves downstream renal parenchymal lesions that cannot be improved by revascularization.¹³ A trial comparing the effects of medication alone (including an angiotensin II receptor antagonist) and medication plus renal artery stenting on cardiovascular outcomes is currently underway.¹⁴

Stenting for ARD does not improve BP and kidney function better than medical treatment. Besides, it is associated with frequent complications. Thirty-one of the 226 stented patients (13.7%) in the trials summarized in the TABLE suffered major complications as defined by current criteria.¹⁵ Stable patients with ARD should be treated first with medical management.¹⁶ Available trials did not include unstable patients with uncontrollable hypertension or with pulmonary edema. It is therefore possible, yet unproved, that renal artery stenting is useful in patients with ARD and refractory hypertension or heart failure, or that it is preferable to abstention in ARD patients given a renin-angiotensin antagonist. With or without revascularization, medical therapy using hypolipidemic and antiplatelet agents and renin-angiotensin antagonists⁷ is required for the prevention of renal and cardiovascular events in patients with ARD.

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First author	Main selection criteria	Stenting procedure (number of patients)	Control (number of patients)	Patients with major complications stent:control	BP at follow-up, mmHg stent:control	Renal function at follow-up stent:control
Van de Ven ⁸ , 1999	uni- or bilateral stenosis ≥50% plus lateralized renography or a rise in Cr ≥20% on ACEI	stent alone (42)	angioplasty alone (42)	10:10	160/90:165/90	Cr 140:134
Cooper ⁹ , 2008	uni- or bilateral stenosis ≥50%	stent + abciximab (25) / stent + protection device (22) / stent + both (25)	stent alone (28)	1/1/3:3	NR	58/52/54:52
Balzer ¹⁰ , 2009	uni- or bilateral ostial stenosis ≥70%	stent alone (22)	surgery (27)	3:2	NR	NR
Bax ¹¹ , 2009	uni- or bilateral stenosis ≥50%, GFR 15–80 ml/min, stable BP	stent alone (62)	medication (74)	10:0	151/77:155/79	10:16 patients with endpoint ^a GFR 50:46

a The primary endpoint was the percentage of patients with a 20% or greater decrease in GFR during a 2-year follow-up.

Abbreviations: ACEI – angiotensin-converting enzyme inhibitor, BP – blood pressure, Cr – creatinine (µmol/I), GFR – glomerular filtration rate (ml/min), NR – not reported

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