Case
A 63-year-old man was referred for progressive hypertension and previously identified, incidental renal artery stenosis. Three years earlier, he underwent a 4-vessel coronary artery bypass (CABG). A year later, recurrent chest symptoms led to repeat coronary angiography, which identified patent bypass grafts except for one vein-graft occlusion. No coronary interventional procedures were undertaken. During catheter withdrawal, an aortogram revealed bilateral renal artery stenosis estimated to be “greater than 70% luminal stenosis.” Serum creatinine was 1.3 mg/dL. Blood pressure was 120/70 mm Hg. Medications consisted of daily administration of atenolol 50 mg, atorvastatin 10 mg, and aspirin 81 mg. There was no other history of atherosclerotic events, including stroke or peripheral vascular disease. No renal interventional procedures were undertaken.

Three months before the present referral, this man developed exertional chest discomfort after walking 6 blocks and several brief episodes of supraventricular tachycardia. These events terminated after adenosine administration during emergency room visits. Blood pressures were between 180 and 200/90 mm Hg, for which amlodipine was added to the patient’s regimen. Thallium stress testing demonstrated a small apical area of ischemia. He was offered radio-frequency ablation therapy for nodal re-entry tachycardia.

Hypertension consultation was obtained. The patient described recent awareness of head discomfort, particularly during the morning hours. Home blood pressure readings ranged between 155/90 and 170/95 mm Hg, whereas readings had been in the 125/70 mm Hg range before. He had discontinued smoking 5 years earlier. Office blood pressures now were 205/108 mm Hg. Arteriovenous nicking, including stroke or peripheral vascular disease. No renal interventional procedures were undertaken.

Discussion
This patient is an individual with minimal hypertension, who was found incidentally during a coronary angiogram to have bilateral renal artery stenosis (RAS). No renal artery intervention was undertaken at that time. Nearly 2 years later, he developed accelerating renovascular hypertension, which improved after endovascular renal revascularization. Kidney function did not change, and the patient continued to need antihypertensive therapy after successful renal revascularization.

Let me emphasize from the outset that management of renal artery disease remains highly controversial. This particular patient illustrates several important dilemmas facing the hypertension consultant regarding management of renal artery stenosis (RAS). The first is how to interpret “incidental” renal artery stenosis detected during vascular imaging for other reasons. The second is how best to manage hypertension that is becoming more resistant to therapy in a patient with known renovascular disease. The third is deciding when to undertake renal revascularization in a patient with this condition. Each of these issues represents an area in which reasonable physicians commonly disagree. They highlight further the difficulty of applying group averages from observational studies and treatment trials to individual patients with unique comorbid disease risks. My objective is to discuss the current status of the data available as they apply to this patient.

“Incidental” Renal Artery Stenosis
Imaging the renal arteries now can be achieved by a variety of methods, including contrast angiography, renal artery doppler ultrasound, magnetic resonance (MR) angiography,
One unplanned result of applying newer methods is the identification of renal artery disease in patients with normal blood pressures. These methods may identify nonfunctional renal artery disease in patients with essential hypertension. Much of the established literature and experience with renovascular hypertension derive from studies undertaken in patients with severe hypertension with the objective of identifying potentially “surgically curable” forms of hypertension. Such cases represent a small subset of all patients with hypertension. As a result, many of the earlier inferences regarding risks and benefits of renal revascularization from older studies must be viewed with caution, particularly since the introduction of endovascular techniques and more effective antihypertensive therapy.

In recent years, reports have appeared from studies using screening aortography as part of the angiographic study of coronary arteries as in this case, abdominal aorta, and peripheral vasculature. Results of these studies indicate that atherosclerotic vascular disease in coronary and peripheral vascular beds commonly is associated with renal artery disease. A recent series identified stenosis of more than 50% of the lumen in 19% of hypertensive individuals during coronary angiography. These lesions exceeded 70% stenosis in 7% of subjects and were occasionally bilateral. When lower extremity symptoms or aortic disease is the primary clinical indication for angiography, renal artery lesions may be seen in up to 50% of subjects. These data underscore the fact that atherosclerotic RAS is common. Its prevalence increases with advancing age in Western societies. It bears mentioning that similar observations were made decades ago based on postmortem pathologic examination of the vasculature before invasive angiography was commonly performed.

What is the clinical significance of detecting renal artery lesions when they are otherwise unsuspected? This question has been the subject of controversy in recent nephrologic and cardiovascular debates, particularly since the introduction of endovascular stents. Some authors argue that a substantial number of patients reaching end-stage renal failure have no other apparent explanation and that unsuspected “ischemic nephropathy” may account for 14% to 20% of patients undergoing dialysis. Some argue, therefore, that “open renal arteries are better than closed renal arteries” and that stenoses should be subjected to arterial repair routinely, usually with endovascular stents. Conversely, others argue that RAS is commonly an incidental “bystander” with little effect on renal function. Follow-up studies of incidentally identified lesions managed without revascularization identify remarkably few cases of progression to advanced renal failure. Recognition that interventional procedures in patients with atherosclerotic disease sometimes pose risks and can lead to complications, particularly in patients with renal artery disease, has led to a more conservative approach.

Figure 1. Blood pressures and medications at time points before and after hypertension consultation in a 63-year-old man. A renal aortogram obtained during coronary angiography identified incidental bilateral atherosclerotic renal artery stenoses exceeding 70% in degree. No intervention was undertaken for more than 2 years after detection.

Figure 2. Aortogram illustrating relatively high-grade, bilateral atherosclerotic lesions located at the ostia of the renal arteries in a 63-year-old man. These were originally identified incidentally during coronary angiography at a time when blood pressures were normal during beta-blocker therapy only. Serum creatinine was 1.3 mg/dL. Nearly 2 years after detection, arterial hypertension progressed to 205/108 mm Hg. This was associated with worsening symptoms of angina, although no new coronary lesions were apparent.

Figure 3. Aortogram after placement of renal artery stents demonstrating widely patent vessels in the patient described in Figure 2. Blood pressures become more readily controlled with resolution of chest pain, although antihypertensive therapy continued to be required.
worsen both renal function and hypertension control intensifies the debate.

Implicit in these arguments is the recognition that high-grade vascular occlusion to the kidneys can both accelerate hypertension and threaten the viability of the kidney beyond the stenotic lesion. Obstruction of the vascular lumen produces a measurable fall in either blood flow or trans-stenotic pressure gradient only after 70% to 80% of the lumen is affected.14 Many RAS lesions are thereby identified long before they pose a hemodynamic constraint to blood flow or renal perfusion. Most imaging techniques are hampered by imprecise estimates of lumen obstruction. Nonetheless, many lesions estimated at “50%” stenosis likely have little, if any, clinical effect. This is particularly the case with MR angiography, which tends to overestimate the degree of stenosis based on MR signal disruption.

The patient presented today represents an example of screening for renal artery disease. He was a candidate for early atherosclerotic disease, having had coronary artery bypass surgery before age 60. The angiographic study was directed primarily at his coronary bed, and renal artery lesions were not specifically suspected. Blood pressure was controlled to normal levels during therapy with a beta-blocker alone. Kidney function was stable and close to normal. Despite the presence of relatively high-grade renal artery disease, no hemodynamic consequence suggesting renovascular hypertension or “ischemic nephropathy” was apparent. His situation was that of an individual with anatomic renal arterial stenosis without the clinical syndrome of “ renovascular hypertension.” His physicians considered it unlikely that vascular intervention at that time would offer material benefit. I agree with that assessment. At this stage, the incidental RAS lesions appeared to have little effect.

Progression of Renovascular Occlusive Disease

Central to the issues raised here, however, is the potential of these lesions to become more severe or to activate pressor mechanisms over time. Atherosclerosis is a progressive disorder that eventually can lead to more severe vascular compromise and total occlusion. Arguments for early intervention have been based on the belief that silent progression of up to 40% of renal artery lesions could occur and may be the basis for some patients developing end-stage renal disease.15 As noted above, some patients with no other apparent renal disease require dialytic support for “ischemic nephropathy.” Should detection of high-grade lesions lead to renal revascularization as a preventive measure?

Several recent studies indicate that progression of atherosclerotic renal artery disease occurs at varying rates. A careful prospective cohort study of atherosclerotic RAS indicates that measurable changes in doppler ultrasound flow velocity develop overall in 31% of patients after 3 to 5 years.16 “Progression” in these studies was based on doppler velocities in the renal arteries studied. Predictors included elevated systolic blood pressures and ipsilateral disease. The rates of progression were higher in those with more advanced disease at the first study than in those with minor or no disease at the outset. Progression to total occlusion was unusual in this series and occurred only in 9 of 295 vessels (3%). A recent trial of medical therapy compared with percutaneous transluminal renal angioplasty (PTRA) reported 16% developing occlusion of a renal artery in the medical arm.17 A small subset of prospectively studied individuals had loss of renal volume (“atrophy” more than 1 cm),18 and fewer yet had measurable loss of renal function. Anatomic and hemodynamic progression, therefore, may be distinguished from “clinical progression,” defined as meaningful changes in blood pressure or kidney function. Follow-up studies of patients with severe RAS (>70% stenosis), managed without revascularization for more than 3 years, indicate that less than 10% to 15% actually develop treatment refractory hypertension or declining renal function that warrants intervention.13 This conclusion is consistent with recent long-term studies from Europe in which incidental renal artery lesions were not associated with progressive renal failure at all.6

Taken together, it may be argued that most renal artery lesions do NOT progress rapidly. We suspect that rates of progression may indeed be diminishing with more aggressive medical intervention aimed at control of lipids, withdrawal of tobacco, and intensive blood pressure control.19 As a result, we argue that it is reasonable to manage and follow patients “expectantly” in many cases of RAS if blood pressure and renal function are well maintained. In point of fact, many patients with RAS are never detected, because they remain stable and easily treated for many years (see below).

Medical Therapy of Renovascular Hypertension

The range of mechanisms underlying hypertension in patients with renal artery disease is complex and beyond the scope of this discussion. An extensive body of literature supports the role of activation of the renin-angiotensin system in developing renovascular hypertension.20 It must be recognized that numerous other mechanisms related to sodium homeostasis, activation of the nervous system, and vascular endothelial function are important at various phases of this condition also. Recent studies indicate that alterations in oxidative pathways favor “quenching” of nitric oxide in the development of increased pressor tone.21,22 How and when each of these pathways participate in systemic blood pressure elevation is not fully understood.

The magnitude of the impact related to the introduction of agents blocking the renin-angiotensin system cannot be overstated. These drugs are now commonly used for reasons other than hypertension alone. Both angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are capable of interrupting the actions of angiotensin II, either by receptor blockade or impairment of its generation. In experimental models, blockade of the renin-angiotensin delays or attenuates hypertension in the face of renal artery lesions.20 These agents are more effective in treating renovascular hypertension than most other antihypertensive drugs.23 In recent years, use of these agents has expanded to indications including proteinuric renal disease, congestive cardiac failure, and most importantly, the prevention of cardiovascular disease end-points in high-risk individuals with diabetes and other major risk factors. Hence, it is probable that many, if not most, patients with incidental atherosclerotic RAS are treated medically before being con-
sidered for renal revascularization, commonly with drugs that block the renin-angiotensin system.

Does medical therapy in patients with unidentified RAS predispose to progressive vascular occlusive disease? Some clinicians have proposed that candidates for ACE/ARB therapy be screened for “occult” RAS before therapy. The positive outcomes of patients enrolled in the large congestive heart failure (CHF) trials with ACE inhibitors, where participants number many thousands, have been reassuring in this regard. Although these populations are among the highest risk for including patients with undetected renal artery lesions, less than 1% to 2% develop progressive renal insufficiency or have therapy withdrawn for that reason. Although the fraction of patients developing significant clinical progression is low, the denominator of patients at risk is quite large. Hence, most clinicians do indeed encounter patients with acute renal insufficiency or other syndromes identifying high-grade RAS that merit revascularization during ACE/ARB therapy. It is important to remain vigilant for such patients and to monitor closely both serum creatinine and potassium during initiation of therapy. Nonetheless, the safety of patients treated with ACE inhibitors and the rarity of renal dysfunction despite a high prevalence of subclinical RAS are reassuring.

Because atherosclerosis is gradual in onset, most patients undergo medical therapy for hypertension as a routine part of cardiovascular disease management. Remarkably, current antihypertensive drug therapy is effective in achieving blood pressure control in the majority of individuals with “incidental” renal artery stenosis. The picture is obscured by a lack of definitive data from prospective, randomized trials comparing renal artery stenting with current antihypertensive medications. Several small, randomized, prospective trials compare medical therapy with PTRA. These studies fail to establish clear benefits of angioplasty but must be considered limited in their scope. All were small (the largest had 106 patients). They excluded patients for whom intervention was considered urgent. Stenting was not part of the intervention. Several had appreciable crossover (ie, later assignment to angioplasty after failed medical therapy, in one instance reaching 44% of subjects). When analyzed by “intention to treat,” the crossover patients were included in the medical arm. As a result, many clinicians argue that these data underestimate those subjects for whom renal revascularization offers major benefits. Many interventional radiologists and cardiologists take the position that stenting has become the treatment standard for ostial atherosclerotic RAS. A prospective, randomized study of renal artery stenting demonstrated improved vessel patency compared with angioplasty for ostial lesions. Remarkably, clinical outcomes did not differ between PTRA and stented patients regarding blood pressure and renal functional changes in this study. Some authors argue that technical advances in interventional procedures are changing rapidly and offer the potential for better outcomes.

Despite evident benefit in some patients, it is equally clear that revascularization, either by stenting, angioplasty, or surgery, leads to little or no benefit in others (15% to 20%) regarding blood pressure control. Many forms of functional testing to establish the physiological role of renal artery lesions commonly used in planning major surgical procedures, such as renal vein renin assays, have been deemphasized in the current era of immediate endovascular intervention. Whether or not this is appropriate is debatable. “Cure” of hypertension with no further antihypertensive medication requirement is rare. As a result, renal revascularization should be considered as one step in the process of long-term management of renovascular disease. Medical antihypertensive therapy is necessary in the majority of patients with atherosclerotic renal artery disease, both before and after renal intervention. The patient today falls into the category of one whose blood pressure was substantially improved after stenting but required ongoing medications (Figure 1).

This patient did not have progressive renal insufficiency but developed a syndrome of progressive hypertension over several months. In fact, comparison of the current angiogram with the aortogram 2 years earlier identified no evident anatomic change in the renal artery lesions. It is recognized that determination of the true onset of renovascular hypertension in humans is most often impossible. This case argues that at some point the renal artery lesion(s) reached “critical” dimensions or activated pressor mechanisms leading to major sustained pressure elevations. These pressure elevations were sufficient to induce higher cardiac workloads and symptoms of cardiac ischemia. In this instance, prior knowledge of renal artery stenoses favored prompt intervention with repeat angiography and revascularization.

Opinion as to whether screening aortography should be undertaken as part of coronary procedures varies among medical centers. Some institutions have made it a standard procedure. The additional volume of contrast is minor, and previous studies indicate that little or no incremental risk is related to the aortogram itself. Follow-up studies of incidentally identified disease indicate that the presence of high-grade renal artery stenosis is an independent predictor of long-term survival. Although the question of screening is distinct from the question of endovascular intervention, in practice, the probability of endovascular renal artery procedure is 4-fold higher within 30 days of coronary angiography when cardiologists are doing the procedure. Our own view is that aortography alone to define renal artery disease is justified to define cardiovascular risk in hypertensive patients undergoing coronary artery procedures. The judgment regarding renal artery intervention should be considered separately with more limited indications.

Timing of Renal Revascularization

If there were no risks or costs attributable to renal artery procedures, this would be a far simpler discussion. Unfortunately, the complications of aortic catheterization, including distal embolization and aortic dissection, can be substantial. A summary of 10 stent series identified serious complications in 13% of patients. Many episodes of atheroemboli go undetected clinically, a common feature in nearly all atherosclerotic interventions. Restenosis with renal artery stents remains a formidable issue in 14% to 30% of cases, leading to technical failure and the potential for repeat proce-
dures.\textsuperscript{36,38} Technical advances may reduce both complication and restenosis rates in the future. The current reality, however, is that renal artery procedures offer moderate benefit to some patients at moderate risk. In some unfortunate cases, clinical results including arterial thrombosis or atheroembolic renal disease produce catastrophic deterioration of renal function and blood pressure control.\textsuperscript{19}

At what point does the balance of risks and benefits tilt in favor of restoring the renal circulation? A complete review of the techniques and complications of endovascular and surgical renal artery intervention is beyond the scope of this discussion. A few points do merit emphasis, however. In recent years, the availability of endovascular stents has changed dramatically the frequency with which atherosclerotic lesions of the renal artery are treated by endovascular methods.\textsuperscript{34} Although none are approved specifically for this purpose in the United States, stents achieve effective patency of ostial lesions, which commonly fail using angioplasty alone. The inclination to undertake renal artery procedures varies dramatically by geographic region and by the subspecialty (eg, interventional radiologists compared with cardiologists) responsible for the procedure.\textsuperscript{34} Surgical repair of renal artery lesions has declined substantially and is most often undertaken as part of aortic repair, eg, for aneurysm. Although surgical risks and morbidity are higher in the near term than with endovascular procedures, long-term durability of repair and survival are excellent after surgery.\textsuperscript{39} It must be understood that restenosis rates with stents remain between 14\% to 30\%, and the long-term patency of these lesions is not well known. I am not certain that the patient today received the best treatment for long-term success. Some of my colleagues favor definitive surgical correction especially for younger patients at risk for further atherosclerotic disease.

Timing is a major concern. Among the most consistent predictors of clinical benefit regarding blood pressure control has been duration or acceleration of hypertension.\textsuperscript{40–42} The patient presented today had previously identified RAS but few clinical manifestations at the time. His blood pressure had become a problem sometime within months of this visit, not years. Experimental renal artery lesions sometimes have a limited time for reversibility,\textsuperscript{43} although the applicability of this observation to humans has not been well established. Few criteria reliably predict the potential clinical benefit in a specific patient, although recent studies of poststenotic renal vascular resistance by doppler ultrasound indicate that high-resistive index is a poor prognostic sign.\textsuperscript{44} Knowledge of the presence of bilateral renal artery lesions at the time of onset undoubtedly biased his physicians toward early intervention in this case.

Patient age and future disease risk are further concerns. As more individuals reach advanced age and succumb less regularly to cerebrovascular and coronary artery disease, the opportunity for RAS to reach critical proportions increases. The average age of renal revascularization has thereby risen substantially in recent years.\textsuperscript{45,46} What is the best option for comparatively young individuals, such as the patient described here? This individual has a propensity to develop accelerated atherosclerosis and, therefore, might be at high risk for rapid progression (and possibly recurrent) disease. It may be argued that such patients warrant early, aggressive intervention to avoid major long-term target injury, such as total renal artery occlusion. The clinicians responsible for his care reached this conclusion. During at least early follow-up, which was confirmed for 2 further years, the benefits regarding blood pressure control and symptomatic relief of cardiac complaints were substantial and positive.

It must be emphasized that the management of this patient cannot be considered complete after renal artery stenting. Atherosclerosis remains a clear and present danger for this individual. Current practice favors intense management of lipid risk, blood pressure control, the withholding of tobacco, and risk-factor reduction. Long-term follow-up studies of patients with renal artery stenosis indicate that these lesions represent an independent risk factor for mortality, whether they are treated or not.\textsuperscript{47}

**Summary**

Taken together, this man’s history illustrates one prone to atherosclerotic disease affecting multiple vascular beds at a relatively young age. Renal artery lesions were identified at a time before they produced significant hypertension or renal dysfunction. They were followed as “incidental” renal artery stenosis. Whether screening for RAS should be considered routinely as part of angiographic procedures cannot be determined with the data available today. Forewarning of the presence of high-grade renal artery lesions in this individual allowed prompt intervention 2 years later when arterial hypertension progressed rapidly and aggravated symptoms of cardiac ischemia. Awareness of the time sequence and potential for progression of both anatomic and clinical manifestations of renal artery disease is important for clinicians caring for such patients, particularly during an era of rapidly changing imaging, medical therapy, and interventional techniques.

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Progressive Hypertension in a Patient With "Incidental" Renal Artery Stenosis

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