Clinical Conference

Accelerated Hypertension in a 64-Year-Old White Woman

Principal Discussant

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Case Presentation

A 64-year-old white woman was referred to the Hypertension Unit of the Massachusetts General Hospital for evaluation and management of resistant hypertension. Past history revealed that she had had mild hypertension for 20 years, adult onset diabetes for 2 years, a myocardial infarction 7 years earlier, congestive heart failure, and angina. Her main complaint at the time of her initial evaluation was dyspnea, which mainly occurred with exertion. Indeed, she could only walk one block before having to stop because of dyspnea. She denied symptoms suggestive of intermittent claudication or cerebrovascular disease, and her angina was stable and mild. Her height was 4 ft 9 in., and she weighed 200 pounds.

The patient’s blood pressure was 196/118 mm Hg standing and 176/122 mm Hg lying, as recorded with an appropriately large cuff. The pulse was 88 bpm and regular. There was no delay of the femoral pulses and no abdominal or arterial bruits. The apex beat was displaced 2 cm to the left of the midclavicular line, and a palpable S4 was evident. Auscultation of the precordium confirmed the presence of a fourth heart sound, but no S3 or murmur was present. Fundoscopy revealed Keith and Wagener (KW) Grade III changes with hemorrhages and marked arteriolar narrowing, but no exudates. The optic discs were sharp, and venous pulsations were evident. Neurological examination was normal. Medications included digoxin 0.25 mg four times daily, isosorbide dinitrate (Isordil) 40 mg four times daily, propranolol (Inderal) 40 mg four times daily, prazosin 6 mg times daily, trinitroglycerin one tablet as needed, hydrochlorothiazide 25 mg/triamterene 50 mg (Dyazide) two tablets four times daily, and isophane (NPH) insulin, 58 U subcutaneously once per day.

The patient was immediately hospitalized, and nifedipine 10 mg four times daily was added to the regimen. Shortly thereafter, she developed overt cardiac failure associated with a drop in blood pressure to 100/90 mm Hg. She responded to conventional therapy and temporary discontinuation of the antihypertensive medications.

Relevant investigations performed over the next week were as follows. An electrocardiogram showed normal sinus rhythm, with changes consistent with an old anterolateral apical myocardial infarction, and ST segment elevation in leads V2-V5 that suggested an anteropapical aneurysm. Renal function, complete blood count, and biochemical profile were essentially normal. Serial cardiac enzyme determinations were normal. The blood sugar level was 283 mg/dl. A gated blood pool scan revealed a markedly hypertrophied left ventricle, a large apical aneurysm that emptied almost completely during systole and an ejection fraction of 54%. Doppler studies of the carotid arteries were normal. Pulmonary function studies showed changes consistent with poorly reversible chronic obstructive pulmonary disease. A thallium scan with persantin provocation was negative for myocardial ischemia. A renal angiogram showed a tight stenosis (greater than 90%) 1 cm distal to the origin of the left renal artery. A renal scan revealed a small left kidney and delayed and diminished perfusion bilaterally, although slightly worse on the left. Renal vein renin concentrations (ng/ml/hr) were as follows: left renal vein 14.7; right renal vein 7.3; inferior vena cava (IVC) above the renal veins 7.0; IVC below the renal veins 7.2.

Eight hours after this procedure the patient developed a deep venous thrombosis of the right popliteal and femoral veins, as confirmed by venography. She was treated with continuous intravenous heparin. With
the diagnosis of unilateral renal artery stenosis, captopril, 25 mg was commenced three times daily and increased to 75 mg three times daily. However, the blood pressure remained only moderately well controlled with values ranging from 180/110 mm Hg to 160/95 mm Hg.

Ten days after admission, a percutaneous transluminal angioplasty was performed on the left renal artery lesion. The blood pressure fell almost immediately to 100/60 mm Hg. All antihypertensive medications were discontinued. The blood pressure rose somewhat over the next few days, but was readily controlled with atenolol 50 mg four times daily and hydrochlorothiazide 25 mg four times daily. Despite this marked improvement, a repeat renal scan was unchanged. The patient was discharged without further sequelae 9 days after the angioplasty.

**Case Discussion**

This case illustrates many important basic facets of the management of severe hypertension, including the decision-making that is involved when renovascular disease is discovered or suspected.

**Aims of the Clinical and Laboratory Assessments**

The aims of the clinical and laboratory assessments in hypertension are to determine the degree of organ impairment, to detect other cardiovascular risk factors coexisting with the hypertension, to discover any potentially curable cause for hypertension, and to identify features that may be important in the choice of antihypertensive drug therapy. These aims are a convenient starting point for our discussion.

**Evidence of Organ Impairment**

**Cardiovascular Impairment**

The history of myocardial infarction 7 years earlier was substantiated by the electrocardiographic findings. There were features of an old anterolateral apical myocardial infarction and an anteropical aneurysm. Cardiac enlargement was evident as a clinical finding and was confirmed by a gated blood pool scan showing a markedly hypertrophic left ventricle with a large apical aneurysm. Severe exertional dyspnea was present, as well as mild angina. The dyspnea was probably due in part to heart failure, although the cardiac ejection fraction was 54%. However, pulmonary function studies also revealed evidence of chronic obstructive airway disease. A coronary angiogram was not performed, but any gross coronary insufficiency should have been detected by the thallium scan.

There was no clinical evidence of cerebrovascular or peripheral vascular disease. However, the retinal arterioles were narrowed, and hemorrhages were present. The presence of hemorrhages on fundoscopy in the patient with severe hypertension is a crucial finding. If retinal vascular occlusion or diabetic retinopathy can be excluded, then malignant hypertension is imminent or already present. The older definition of malignant hypertension required the presence of papilledema, but many authorities now accept the presence of hemorrhagic and/or exudative retinopathy as being sufficient for this designation. Apart from the risk that a hemorrhage may occur in the region of the macula, the finding of malignant (KW III or IV) hypertensive retinopathy implies that fibrinoid necrosis may be present in the arterioles of the kidney. Parenthetically, it should be noted that in this case one kidney was protected from the hypertension by the stenosis in its artery.

Adult onset diabetes mellitus is not generally associated with severe diabetic retinopathy. Presumably, we can accept the judgment of the clinician who labeled the changes as KW III, that is, the hemorrhages were due to hypertension rather than to diabetes.

**Renal Impairment**

The clinical presentation did not suggest the presence of kidney disease or of renal functional impairment. In the past, it used to be taught that all patients presenting with hypertension should have an intravenous pyelogram performed. However, the follow-up of a large series of screening tests in hypertension has suggested that routine pyelography is infrequently positive and cannot be considered cost-effective.

Moreover, occasional severe and even fatal anaphylactic reactions occur from the intravenous injection of contrast medium, and acute tubular necrosis is a rare but important complication, particularly in diabetics and the elderly. Thus, we usually confine pyelography to those hypertensive patients presenting with a history or clinical findings suggestive of past or present renal disease.

**Coexisting Cardiovascular Risk Factors**

The principal aim of management in hypertension is to arrest the progress of cardiovascular disease. It is pointless to treat the hypertension without recognizing and attempting to treat other cardiovascular risk factors. Diabetes mellitus and obesity are such factors in this case. Hypercholesterolemia and hypertriglyceridemia should be excluded. Irrespective of the results of the blood lipid analysis, it is not surprising to learn that vascular attrition had occurred in this postmenopausal woman. We are not told whether the left renal artery stenosis was due to fibromuscular dysplasia (probably congenital in origin) or to an atheromatous lesion. The latter is far more likely in this case, however.

Another risk factor that would be carefully evaluated during history taking in Australia would be analgesic abuse. Long-standing overuse of compound analgesics that contain aspirin and phenacetin has been strongly associated in Australia and other countries with accelerated development of vascular disease, particularly atheromatous renal artery occlusion. Other preventable cardiovascular risk factors, which apparently were not present here, include smoking and possibly a high sodium intake.

**Potentially Curable Causes of Hypertension**

Important diagnoses to be excluded in every new patient with established hypertension are coarctation...
of the aorta, pheochromocytoma, Conn’s syndrome due to an adrenal adenoma, and renal artery stenosis. Coarctation was ruled out by finding no delay in the femoral pulses. There were no paroxysmal symptoms to suggest a pheochromocytoma, although it probably would have been worthwhile to exclude this diagnosis more fully by measuring urinary catecholamines. There were no symptoms such as nocturia or muscle weakness to suggest Conn’s syndrome, nor was the serum potassium decreased. Had peripheral plasma renin studies been carried out, they might have shown some elevation of plasma renin during the period when the patient was off antihypertensive medications. Such a finding would have also tended to exclude primary aldosteronism. Hyperreninemia and secondary hyperaldosteronism are usually found in unilateral renal artery stenosis and would have been exaggerated in this case during the period of overt cardiac failure. Apparently, the decision to proceed with the renal angiogram, which revealed the underlying lesion, was taken on general clinical grounds and because scintiscanning revealed a small left kidney. Indications for renal angiography will be discussed further below.

Clinical Points Assisting Choice of Drug Therapy

From the outset of the clinical examination, cognition should be taken of factors that direct the choice of antihypertensive drug therapy. Examples of such factors are given in Table 1. It is evident that the choice of the beta-adrenergic-receptor blocking drug Inderal (propranolol) was probably unfortunate here. Beta-blockers are contraindicated for use in patients with congestive cardiac failure and are generally unsuitable when there is chronic obstructive airway disease or unstable, insulin-requiring diabetes mellitus. The choice of captopril for treating the hypertension, once the diagnosis of renal artery stenosis was made, is of interest. Captopril was initially hailed as the drug of choice in this condition. Captopril is a converting-enzyme inhibitor and interferes with the function of the renin-angiotensin system by preventing the formation of the vasoconstrictor octapeptide angiotensin II. Since the hypertension of unilateral renal artery stenosis is angiotensin-dependent, captopril should specifically counter the pathogenesis of the disorder. However, it became apparent that suppression of the renin-angiotensin system in renal artery stenosis might not always be beneficial. Indeed, in some patients with bilateral renal artery stenosis or with renal transplant renal arterial stenosis, a steep decline in renal function has been observed after captopril. The basis of this effect is fascinating. It seems that the ischemic kidney is dependent upon renin release to cause angiotensin-mediated constriction of the postglomerular circulation. This preserves its glomerular-filtering function in the face of a diminished head of arterial pressure. Captopril prevents this effect and thereby reduces the glomerular filtration rate and overall renal function. In this case, we are given no evidence of deteriorating renal function. Presumably, the contralateral kidney was able to sustain a satisfactory level of renal function. However, the blood pressure was not well controlled. The advantages of captopril in reducing circulating angiotensin levels may have been countered by fluid retention.

Renal Angiography

Factors in Deciding Whether to Proceed to Angiography

Renal angiography is the only definitive way to reach a diagnosis of renal artery stenosis. Unfortunately, less invasive screening methods for renovascular disease are associated with an appreciable incidence of false negative results. The method longest employed is rapid-sequence intravenous pyelography. In a recent retrospective study in 92 patients with renal artery constriction, intravenous pyelograms were found to be normal in 31. In 181 patients from the same study with normal angiograms, the intravenous pyelogram was found to be abnormal in 32. Thus, as an index of the diagnosis of renal artery abnormalities, the intravenous pyelogram was found to give a 33% false-negative and a 17% false-positive rate. Better predictive results have been reported from other studies. However, with the advent of less invasive techniques of angiography, the intravenous pyelogram is likely to become obsolete as a method of screening for renal artery stenosis. Newer computer technology, isotopic renal scans are becoming more popular in some centers as a method of screening for renal artery stenosis. Techniques such as renal vein renin estimation and divided renal function studies are too unreliable and elaborate for use at the screening stage.

Given that the incidence of renovascular disease is probably less than 0.2% among hypertensive patients, and increasingly larger numbers of people with milder hypertension are coming under scrutiny as subjects at risk for hypertensive cardiovascular disease, which patients are to have renal angiography? This question is best approached by considering the decision-making process faced at two phases in the patient’s management history.
Phase 1: The Newly Detected Hypertensive Patient

During the initial evaluation of the newly detected hypertensive patient, a careful search must be made for features in the history and clinical examination that increase the likelihood of renal artery stenosis. The lack of any history of hypertension or stroke among first-degree relatives in a young person with hypertension is suggestive. At one stage it used to be taught that all young people presenting with hypertension should be screened for renal artery stenosis because it was unlikely that they could have essential hypertension. However, recent experience indicates that renal arteriography is unlikely to provide useful information in patients under 40 years of age. Of 152 patients under 40 years of age who had well-controlled blood pressure, normal physical examinations, normal renal function, and normal intravenous pyelograms, 138 had normal renal angiograms, 12 had minor narrowing of the renal arteries of no hemodynamic significance, and two had renal artery stenosis, only one case of which was suitable for surgical repair.

The presence of an epigastric bruit is an important sign of renal artery stenosis, particularly in young hypertensive patients. No bruit was heard in the present case, but this may have been due to her extreme obesity. Renal artery bruits are best heard by pressing lightly just above the umbilicus. Care must be taken not to produce artifacts by heavy pressure on the aorta in lean subjects.

The finding of impaired renal function in a hypertensive patient without preexisting renal disease is a reason to consider bilateral renal artery stenosis. A plain x-ray of the soft tissues of the abdomen, combined with tomography if required, is a worthwhile measure in any newly detected hypertensive patient. The discovery of a significant disparity in renal size, that is, the right kidney more than 2 cm shorter than the left or the left kidney more than 1 cm shorter than the right would signal the need for further investigation. The cause is most likely to prove either unilateral ureteric reflux or renal artery stenosis.

Phase 2: The Patient Who Has Proved Refractory to Antihypertensive Therapy

If the blood pressure is not satisfactorily controlled by conventional antihypertensive drugs, or if renal function should deteriorate despite attempts at blood pressure control, it is time to review the possibility that renal artery stenosis has been missed. The balance between the risk of renal angiography and that of undiagnosed renal artery stenosis has been shifted toward performing angiography. In the present case, there were many reasons for investigating the possibility of a correctable underlying cause for the hypertension. There was gross organ impairment and uncontrolled hypertension, despite the use of combined alpha- and beta-adrenergic-receptor blockade. The patient’s diabetic and cardiac status posed difficulties for the use of beta-blockers or diuretics in treating the hypertension. The presence of a left ventricular aneurysm was a particular threat because of the risk of embolization, particularly if the hypertension remained uncontrolled. Thus, a decision was made to perform a renal angiogram despite the lack of specific evidence for renal artery stenosis.

Newer Techniques

In discussing the trade-off between the risks and benefits of renal angiography, it is important to consider briefly the significance of newer methods of angiography that are becoming more widely available. Intrarrenal, digital subtraction angiography (DSA) offers an advance over conventional angiography by improving contrast resolution. Smaller volumes of a less viscous contrast agent are required. The catheters introduced into the aorta and renal arteries via the femoral artery can be of narrower caliber and can result in less trauma to the femoral artery wall. There is less chance of bleeding from the puncture site after the procedure or of femoral artery aneurysm. It may be possible to perform the procedure on an outpatient basis. Intravenous renal DSA offers the hope of reducing the invasiveness of angiography even further. However, this technique may never be sufficiently reliable for definitive diagnosis. The most recent technological advancement is nuclear magnetic resonance (NMR), yet to be evaluated fully in renal angiography. However, the cost of the plant required for this procedure will exclude its widespread use for some years.

Renal Artery Stenosis

Functional Significance

Apart from an impression of unilateral hypoperfusion gained from rapid-sequence intravenous pyelography or from isotopic renal scans, arteriographic evidence that the presence of unilateral renal artery stenosis is causally linked to the subject’s hypertension has been sought from the divided renal function test (Howard-Stamey), from the blood pressure response to the administration of angiotensin inhibitors, and from renal vein renin measurement.

In the present case, three lines of evidence were available. Firstly, the renal scan showed delayed and diminished perfusion which was worse on the left. However, relief of the ischemia of the left kidney by angiotoplasty did not alter the scan. Secondly, there was an equivocal blood pressure response to the angiotensin inhibitor captopril. Thirdly, the renal vein renin ratio was 2.0, with lateralization to the left; this implies a 95% likelihood of successful relief of hypertension from revascularization. A definitive result was obtained only from the third procedure done here highlights its preeminent role as a prognostic test.

It must be emphasized, however, that the test is only reliable for predicting success, and not for predicting failure. Thus, in a recently reported series of 18 patients with successful outcomes from surgery, 11 (61%) had renal vein renin ratios less than 2.0, and seven (39%) had ratios less than 1.5. According to which criterion was used, either 61% or 39% of these patients would have been excluded from surgery if reliance had been placed upon this test alone.
In contrast with this report is our own experience in determining the renal vein renin ratios after diazoxide stimulation.** We have reported four cases, one with unilateral renal artery stenosis and three with bilateral stenoses, whose renal vein renin ratios were between 0.8 and 1.3 prior to stimulation of renin release, but whose ratios rose to values between 1.8 and 4.7 after administration of diazoxide, 300 mg intravenously.** Each of these patients had significant improvement in hypertension after subsequent corrective surgery. Thus, inadequate technique may explain some apparently false-negative renal vein renin ratios.

**Natural History and Prognosis with Therapy**

Before discussing various approaches to the treatment of renal artery stenosis, it is important to review what is known about the natural history of this condition. Schrieber et al.** showed that atherosclerotic stenosis of the renal artery progressed in 37 (45%) of 85 patients. In 14 of the 37, the lesion went on to total occlusion and shrinkage of the kidney. Progressive lesions were found in 33% of the patients with fibromuscular dysplasia. Patients with bilateral renal artery stenosis may present in acute renal failure.

In attempting to analyze prognosis with therapy, it is helpful to consider patient mortality, control of blood pressure, and effects on renal function. In a 1968 study of 69 unoperated, medically treated patients followed for 1 to 6 years, mortality was 40%.** In the same study, 35% of 43 patients with renal artery stenosis treated by vascular bypass or unilateral nephrectomy were dead after the same observation period. Other studies** have demonstrated a better survival after corrective surgery. Thus, after a follow-up period of 7 to 14 years, the survival in 100 patients treated with antihypertensive drugs was 66%, compared to a survival of 84% in 114 similar patients treated operatively.** In the same study, 93% of the patients alive in the operated group were judged to be cured or significantly improved in regard to their hypertension. It has been shown that there is a direct relationship between success in blood pressure control and duration of patient survival. Also, it is clear that short-term survival is greater in patients under 40 years of age.

The literature suggests that about 60% of the patients subjected to corrective surgery for renal artery stenosis may be expected to have good blood pressure control as a result. Revascularization of the ischemic kidney may also be expected to improve renal function.** In the case of patients with renal artery stenosis on one or both sides, and with end-stage renal failure, there have been reports of dramatic improvement after revascularization of occluded arteries.

There is limited information on the long-term follow-up of patients in whom renal artery stenosis has been corrected by percutaneous transluminal renal angioplasty.** The morbidity and mortality of this procedure are less than that of surgical therapy. Emergency surgical repair for abortive attempts at balloon dilation, which resulted in rupture, is required in 1% of cases.** Short-term remission or improvement of hypertension after renal angioplasty (at 1 year) is said to occur in approximately 75% of the patients with fibromuscular disease and in 50% to 60% of the patients with atheromatous disease.** However, in a more recent report of 89 patients followed for an average of 16 months, blood pressure levels were reduced to normal or improved in 93% of the patients with fibromuscular dysplasia and in 84% with atheromatous disease.** Angiographic follow-up at 2 years in 15 patients showed persisting relief of the stenoses and a 12% increase in kidney size.

Angioplasty seems best applied to short, partially stenotic lesions that are completely within the renal artery. Atheromatous lesions located in the renal artery ostium are not amenable to angioplasty, and the procedure is rarely successful when there is complete or near complete occlusion of the renal artery, even if a guidewire can be introduced.

**Selection of Therapy in the Present Case**

This case demonstrates the important therapeutic advance made possible by the technique of renal angioplasty in the treatment of renal artery stenosis. This patient belongs to a group that shows the poorest response rates to renal vascular bypass surgery. General contraindications to surgery in her case consisted of uncontrolled diabetes mellitus, severe obesity, severe myocardial disease, and chronic obstructive airway disease. Her risk factors and age would have probably excluded her from corrective surgery or even nephrectomy in most centers. Before the advent of angioplasty, she would have been consigned to a course of more strenuous medical therapy, which may have proved ineffective. Already, a trial of captopril had succeeded only in controlling her diastolic blood pressure to a level between 95 and 110 mm Hg. Minoxidil, as an alternative potent antihypertensive drug, may have been worth a trial, but would probably have provoked hypertrichosis, fluid retention, and an unfavorable cardiac response.

Perhaps it is worth repeating earlier comments about beta-blockers, in view of the choice of therapy to control her residual hypertension. Although atenolol is a beta,-selective adrenergic receptor blocker, it has been my observation that this drug is not exempt from the respiratory side effects that limit the use of the nonselective beta-blockers in patients with chronic obstructive airway disease or asthma. Even if this patient were free of serious respiratory symptoms on atenolol, I would fear that complications might occur due to bronchospasm were she to contract an acute respiratory infection.

**Concluding Remarks**

In summary, I have endeavored to discuss this case in the setting of clinical hypertension in its broader aspects and to deal with some specialized features of topical interest in the management of hypertension due to renovascular disease. In 1984, there are increased incentives for looking assiduously for undetected renal artery stenosis in our hypertension clinics. We have...
improved methods of angiography that are less invasive and safer than the older techniques, and we have a means of nonsurgical repair of constricted renal arteries. Additionally, the growth of renal transplant units has greatly improved the resources at hand for vascular bypass surgery, when this is required. These advances have tended to decrease the emphasis on utilizing renal vein renin estimation and other lateralizing techniques in reaching a decision whether or not to attempt repair. However, there is more reason than ever to screen carefully for epigastric bruits and to be alert for the possibility of renal artery stenosis in patients with refractory hypertension. There will be continued interest in the possible development of newer radiographic scanning techniques and newer radiographic methods for noninvasive diagnosis of renal arterial disease.

Questions and Answers

Dr. Edgar Haber (Cardiac Unit, Massachusetts General Hospital): I have both a comment and a question. The comment relates to your discussion of approaches to stimulating renin production prior to measurement of renal vein renins. You might recall that my colleagues and I published a paper a number of years ago in which we employed teprotide, a peptide-converting-enzyme inhibitor, as the stimulating agent. We picked a group of patients strongly suspected of having renal artery stenosis, but who had equivocal differences between unstimulated renal vein renins on either side. After stimulation, there was no question about lateralization in those patients ultimately demonstrating unilateral renal artery stenosis. Ratios of renin activity between the two sides were as high as 6, or 8:1. However, those patients who ultimately proved not to have unilateral renal artery stenosis did not change the close ratio demonstrated initially in the unstimulated state. I believe that stimulation should be an essential part of every renal artery renin measurement. There are now a number of good choices among renin-stimulating agents, and I am not sure that one of these agents has significant advantages.

The question is directed at your discussion of outcomes following the correction of renal artery stenosis. Why is there often only a partial amelioration of hypertension, as in the patient discussed today, even though the gradient across the stenosis may be fully relieved?

Dr. Gordon Stokes: In response to your comment, the utility of renal vein renin stimulation was the subject of an editorial a few years back in which Dr. MacCarthy and I pointed out that renal vein renin assessment really must be done in the presence of adequate stimulation. Our findings resembled your experience with the effects of teprotide stimulation in sorting out these problems. I agree also that it doesn’t really matter which stimulants you use, but I think that some are better than others.

Turning to your question, I think that many issues enter into consideration here. I really don’t know what the condition of the small ipsilateral kidney was in the present case. Since the renal vein renin values were lateralized with a 2:1 ratio, correction of the stenosis should have resulted in complete amelioration of the hypertension. However, persistent hypertension after correction of renal artery stenosis has been described in such cases and generally reflects a role of the contralateral kidney in this sustained blood pressure elevation. Indeed, in the past, nephrectomy of the contralateral kidney has been performed in such cases and was not infrequently successful in ameliorating the hypertension. I suppose the other possibility is that this patient had genetically induced hypertension or some other undetectable factor that was independently maintaining the elevated pressure. Finally, the blood pressure may have remained elevated because of structural vascular changes.

Dr. Robert M. Graham (Cardiac Unit, Massachusetts General Hospital): There were two other diagnoses that I entertained in this patient, which Dr. Stokes may like to comment on. One was a dissection of the aorta, which could certainly lead to this sort of picture, although usually the presentation is more acute. Such patients do not always present with a sudden onset of chest pain. The second diagnosis, particularly where this patient was known to have a large ventricular aneurysm, was the possibility of renal artery embolism from a mural thrombus.

Dr. Stokes: I think that any patient who presents with central abdominal pain, particularly with evidence of extensive atheroma, has to be considered a candidate for dissection of the aorta. Of course, this can easily strip up the renal arteries and result in a situation where you get uncontrolled hypertension.

Intrarenal arterial embolization is one of the feared complications when renal artery stenosis remains untreated for a period. We have actually seen a patient with widespread atheroma and bilateral renal artery stenosis whose surgery was deferred. When she did come to surgery after an acute presentation with renal failure, there was evidence of infarction in both kidneys due to extensive emboli. This was a very dramatic example of what can happen. So I agree that these diagnoses should be seriously considered.

Dr. Garner Haupert (Renal Unit, Massachusetts General Hospital): Do you feel that there is any role for stimulated peripheral renin determinations, such as following intravenous furosemide, if one seriously suspects that the patient has renin-dependent hypertension?

Dr. Stokes: I’m doubtful about the value of peripheral renin determinations as a diagnostic measure. As I pointed out in this presentation, sometimes they can be helpful as a guideline to further investigation. However, I think that there are too many problems with using this test as a basis for primary screening of renovascular hypertension. If you use peripheral renin stimulation at all in the diagnosis, I guess it’s reasonable to use some kind of standardized stimu-
lation. Many have been suggested, such as diuretics or a low sodium diet. Again, as Dr. Haber mentioned, it really doesn’t matter too much what type of stimulation you use. By contrast, in the case of renal vein renins, I think that renin stimulation greatly enhances the sensitivity and specificity of the test.

References

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