PARKLAND HOSPITAL MEDICAL GRAND ROUNDS

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AN ELDERLY WOMAN WITH SEVERE HYPERTENSION AND RENAL INSUFFICIENCY

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HISTORY OF PRESENT ILLNESS

A 76 y/o WOMAN with a history of coronary artery disease, hypercholesterolemia, and hypertension with a mildly elevated serum creatinine of 1.8 mg/DL. The patient was in good health after a triple coronary artery by-pass graft operation in April, 1990. In February, 1991, the patient presented to her internist with a history of headache and shortness of breath. She was found to be hypertensive and in mild congestive heart failure. At that time her diastolic blood pressure was between 100-110 mm/Hg. She was on anti-hypertensive medication which included long-acting nifedipine 90 mg/day and atenolol 50 mg/day. The atenolol was discontinued and due to the hypertension and congestive heart failure furosemide was initiated at 40 mg/day. At that time, her BUN was noted to be 38 mg/dl and her serum creatinine 1.8 mg/dl. Four days later, the patient was again seen by her physician and enalapril 5 mg/day was initiated. Blood tests, which were drawn but the results of which were not available until after enalapril was begun, showed a BUN of 61 mg/% and a creatinine of 5.8 mg/dl on 2-18-91. The patient was contacted and instructed to return for admission to Vanderbilt University Medical Center. Enalapril was also discontinued at that time. On admission, the patient was relatively asymptomatic having had significant improvement in her symptoms of shortness of breath. She did note that ever

since the furosemide was started, the volume of her urine output had increased . Her weight, which was 173 pounds on 2-14-91, was 167 pounds on admission. The patient denied taking any medications other than those prescribed and, specifically, denied ingestion of any prescription or non-prescription non-steroidal anti-inflammatory drugs.

Her significant past history included coronary artery by-pass surgery in 1972 with repeat surgery in 1990 for recurrence of disease. She is known to have had hypercholesterolemia for a number of years and also has a 30 pack-year history of cigarette smoking. The patient also was known to be hypertensive for several years prior to admission. In February, 1989, her serum creatinine was 1.3 mg/dl and in February, 1991, was 1.8 mg/dl. With the exception of her hypertension and heart disease, she has been in general good health.

PHYSICAL EXAMINATION

Physical examination on admission showed her to be afebrile with a blood pressure of 146/82 mm Hg, pulse 72 per min, and a weight of 167 pounds. Her examination was within normal limits with the following exceptions: a few bi-basilar rales on chest exam, an S4 gallop, a left upper

quadrant bruit and bilateral femoral bruits.

LABORATORY EVALUATION

Initial laboratory evaluation showed serum electrolytes in mEq/l:sodium 137, potassium 5.1, chloride 104, CO_2 20. Her blood glucose was 197 mg/dl, blood urea nitrogen was 97 mg/dl and serum creatinine 8.1 mg/dl. Uric acid was 11.3 mg/dl, calcium was 9.3 mg/dl, and phosphorus was 5.7 mg/dl. One week prior to admission, the uric acid was 7.2 mg/dl and the serum calcium and phosphorus were 10.4 and 3.4 mg/dl respectively. Hematocrit was 37% and the white blood cell count, 6,400 per mm^3 with a normal differential. Urinalysis showed a specific gravity of 1.013, pH 5, and 1+ albumin. Urine microscopy demonstrated a few granular casts, occasional red blood cells, many white blood cells, and significant bacteria. Gram stain of the urine showed numerous gram negative rods. On admission post-void residual was determined at 60 cc. Renal ultrasound showed no hydronephrosis. The right kidney was about 9 cm in length and the left kidney approximately 7 cm in length. Chest x-rays showed cardiomegaly and a small left pleural effusion. A fractional excretion of sodium was determined on the first spot urine sample obtained in hospital and was calculated to be 1.6%.

DIFFERENTIAL DIAGNOSIS

On admission it was felt that the patient had acute renal failure. Acute volume contraction was considered since the patient had recently been diuresed, but there was no clinical evidence of significant volume depletion. Also included in the differential diagnosis were: acute interstitial nephritis secondary to furosemide; atheroembolic renal disease with spontaneous severe cholesterol embolization of the kidneys; and acute renal failure due to the ingestion of non-steroidal anti-inflammatory drugs, even though denied by the patient.

Early attempts were made to narrow the differential diagnosis by: (1) examining the urine for eosinophils; (2) repeating the white cell differential counts to look for eosinophilia, coupled with a thorough daily physical examination to look for cutaneous and peripheral manifestations of atheroemboli, i.e. digital ischemia and livedo reticularis; (3) duplex scanning of the arteries was performed to assess renal perfusion. On multiple examinations, neither the white blood cell count nor the percentage of eosinophils became abnormal. Also, there was never any clinical evidence cholesterol emboli. Duplex scanning was "technically inadequate".

Over the next five hospital days, the serum creatinine fell from a peak of 8.4 mg/dl to a nadir of 5.5 mg/dl. Urine output ranged between 750 and 1200 ml/day. Blood pressure, which on admission was almost normal, gradually rose to 180/100 mm Hg while both the furosemide and enalapril were withheld. Because of my continued for underlying ischemic renal disease, the patient underwent an intra-arterial digital subtraction angiogram to evaluate renal perfusion bilaterally. The patient was shown to have total occlusion of the right renal artery at its origin with late reconstitution of the main renal artery from collateral flow. There was near total occlusion of the left renal artery at its origin (Figure 1). A total of 35 ml of contrast material was used. Over the several days post-angiography, the patient maintained a serum creatinine of 5.0 mg/dl but had somewhat difficult to control blood pressure with pressures as high as 200/110 mm/Hg. A surgical approach to correcting the ischemic renal disease was offered to the patient and she agreed to undergo vascular repair. On 3-21-91, after a stormy hospital course that included worsening renal failure during attempts to control her hypertension, subsequent development of pulmonary edema and requirement for dialysis, the patient underwent surgery. At operation the vascular surgeon was able to perform bilateral primary re-anastomose of the renal arteries to the aorta. Before surgery the patient had been oliguric and dialysis dependent. Urine

output, which pre-operatively was in the 300-500 ml/24h range, increased immediately post-operatively to > 100 cc/hr. Also, immediately post-operatively systemic blood pressure was easily maintained in the 130-140/75-85 range with PRN sub-lingual nifedipine and intravenous nitroglycerine. Serum creatinine fell from its pre-operative, pre-dialysis level of 8.6 mg/dl to 2.7 mg/dl 48 hours after surgery. Urine output remained excellent. Blood pressure one week post-op was normal on only low doses of long-acting nifedipine.

DIAGNOSIS: Ischemic Renal Disease

Ischemic renal disease is defined as a clinically significant reduction in glomerular filtration rate in patients with obstruction to renal blood flow. Obstruction of blood flow to the kidney can be responsible not only for ischemic renal disease, but also for the development of a second major clinical syndrome, renovascular hypertension.

ANATOMY

To understand how obstruction to renal blood flow can result in ischemic renal disease, it is helpful to review the anatomic variations that can produce this syndrome (Fig. 2). First, ischemic renal disease can result from unilateral renal artery stenosis/thrombosis involving a solitary kidney (native or transplant). Second, it can result from bilateral renal artery stenosis or thrombosis in a patient who has two kidneys. In the setting of two kidneys, the patient can have total occlusion of both renal arteries; total occlusion of one renal artery (like our patient) and a significant stenosis of the contralateral renal artery; or significant renal artery stenosis bilaterally with hypoperfusion of both kidneys. Lastly, ischemic renal disease can result from

unilateral renal artery stenosis in one of two kidneys when a contralateral kidney is atrophic or nonfunctional. Collateral pathways (Fig. 1) available in the presence of renal artery occlusion include the suprarenovascular system, the lumbar vascular system, and the ureteric vascular system.

EPIDEMIOLOGY AND NATURAL HISTORY OF Ischemic RENAL DISEASE

Ischemic renal disease may result from renal artery obstruction disease secondary to embolism (thromboembolism or cholesterol embolism), Takayasu's arteritis, neurofibromatosis, systemic necrotizing vasculitis, dissection of the aorta or arterial trauma. However, atherosclerotic disease of the renal arteries and fibromuscular dysplasia are the two most common causes of ischemic renal disease (Table 1) (1). In a recent prospective survey over an 18 month period, atherosclerotic renal artery disease was the cause of renal failure in 14% of the patients over the age of 50 years accepted for renal replacement therapy (2).

The prevalence of ischemic renal disease secondary to atherosclerosis can be estimated from the incidence of atherosclerotic renal artery lesions leading to renovascular hypertension and the natural history of these lesions.

Meaney (3) and Wollenweber (4) were the first to report progression of atherosclerotic lesions in the renal arteries (as assessed by serial arteriography) of 36% and 63% respectively of patients studied with a mean interval of 24 months between angiograms. More recently, Dean (1981) analyzed the outcome of 41 patients with atherosclerotic renal vascular hypertension treated medically and found that 41% of these patients required surgery due to deterioration in renal function or a reduction in renal size. Of note, neither hyperlipoproteinemia nor poor blood pressure control correlated with progression of the renovascular disease. Importantly, significant deterioration of renal function occurred in some patients whose blood pressures were documented to be well controlled.

The exact incidence or prevalence of ischemic disease is difficult to estimate since few studies have addressed this point. Atherosclerotic renal artery disease accounts for 60 to 70% of the renal artery lesions and the remainder are largely due to fibromuscular dysplasia (5). One can estimate the potential size of the problem of ischemic renal disease from data on the natural history of atherosclerotic renal disease. It can be assumed that there are approximately 600,000 patients in the United States with renovascular hypertension and that two-thirds of these patients have atherosclerotic renal

artery stenosis. Of these, about 40% are being treated medically and 40% of these medically treated patients will demonstrate progression. One can estimate that a minimum of 5% of the progressors reach end-stage renal disease annually or approximately 3,000 patients in the United States each year. This conservative estimate represents about 1 out of every 10 patients who are initiated annually on dialysis.

CLINICAL PRESENTATIONS

Ischemic renal disease can present with any one of a variety of clinical features summarized in Table 2. Patients may present with acute renal failure. The acute renal failure can develop 1 to 14 days after the initiation of antihypertensive therapy with angiotensin converting enzyme (ACE) inhibitors (6). Typically the acute renal failure is reversible after stopping the ACE inhibitor. There are at least two pathophysiologic contributors to renal failure in this syndrome. The first is hypoperfusion of the kidneys due to a major fall in blood pressure in the setting of critical renal artery stenosis. However, acute renal failure after administration of an ACE inhibitor can also occur without a major reduction in systemic blood pressure due to the unique role angiotensin II plays in intrarenal hemodynamics. In patients with fixed obstruction in the main renal artery, the reduced renal plasma flow would normally be associated with a major reduction in glomerular filtration rate (GFR). However, GFR is preserved by the maintenance of high glomerular capillary pressure as a result of angiotensin II-mediated constriction of the efferent arteriole. When intrarenal angiotensin II generation is blocked by ACE inhibition, efferent arteriolar constriction decreases and glomerular capillary pressure falls. Due to the fixed obstruction in the main renal artery,

there is no compensatory increase in glomerular plasma flow and, hence, the GFR falls precipitously. The incidence of this ACE inhibitor-induced acute renal failure has been reported in between 6% (7), and 38% (8) patients with either bilateral renal artery stenosis or unilateral renal artery stenosis in a solitary kidney. The development of acute renal failure following the initiation of ACE inhibitor therapy should suggest the possibility of underlying ischemic renal disease. The drug stopped, and the patient's renal function should be allowed to recover to baseline.

Patients who receive anti-hypertensive drugs other than ACE inhibitors also can develop acute renal failure in the setting of ischemic renal disease. Indeed, acute renal failure can be precipitated by diuretics, clonidine, beta blockers, alpha-methyldopa, minoxidil, calcium-channel blockers and intravenous sodium nitroprusside.

Ischemic renal disease should also be suspected in individuals who present with progressive azotemia and have a known history of renovascular hypertension. Similarly, patients who have progressive azotemia and hypertension, who also have clinical features suggestive of renovascular hypertension, should be evaluated for ischemic renal disease. Some of the

clues suggestive of renovascular hypertension include: hypertension in the absence of a family history, hypertension presenting at an age < 25 years or > 45 years, abrupt onset of moderate to severe hypertension, malignant hypertension, history of flank pain and hematuria, or presence of flank or abdominal bruit.

Unexplained azotemia in an elderly patient with other evidence of atheromatous disease may be another clinical clue to the presence of ischemic renal disease. 80% of patients with renal artery stenosis have clinical evidence of generalized atherosclerotic disease (9).

Lastly, hypertension and azotemia in a renal transplant patient in whom obstruction, cyclosporine nephrotoxicity and rejection have been ruled out, should suggest the possibility of renal artery stenosis leading to ischemic renal disease.

DIAGNOSTIC PROCEDURES

The diagnosis of ischemic renal disease can be suspected on clinical grounds but cannot be established without specific diagnostic maneuvers.

Although there are some similarities between the evaluation of patients with suspected ischemic renal disease and that of patients with suspected renovascular hypertension, a fundamental difference exists between these two situations. The diagnosis of renovascular hypertension is most commonly associated with at least one normally functioning kidney. The tests employed to diagnose renovascular hypertension take advantage of this point and they involve a comparison of the two kidneys. Clearly, in ischemic renal disease, which by definition involves both of two kidneys or the renal artery of a solitary kidney, none of the tests comparing kidney function are useful. Potential diagnostic tests which can be used to identify ischemic renal disease are listed in Table 3. Although both the rapid sequence IVP and the renal ultrasound will provide anatomical data such as the presence of one or two functioning kidneys or a major discrepancy in renal size, they are not specifically diagnostic for ischemic renal disease. Indeed, the former test is contra-indicated.

Early radionuclide studies of the kidney employing isotopic renal blood flow scans (Tc-99m-labelled DTPA), and renal functional scans (I-131 Hippuran) were unable to improve upon the sensitivity and specificity of the IVP examination. Disorders not involving major arteries, such as chronic

glomerular disease, can produce abnormalities similar to those seen in renal artery stenosis. More recently, reports have suggested that combining these radionuclide techniques with the pharmacologic challenge of ACE inhibition may offer improved sensitivity in detecting renovascular hypertension. Unfortunately, although isotopic renography with the use of captopril may prove very useful as a screening test for renovascular hypertension (10), it is unproven as a diagnostic test in the setting of ischemic renal disease.

More recently, duplex scanning of the renal artery has been proposed as a non-invasive way to diagnose significant renal artery stenosis (11). This technique uses B-mode ultrasound combined with a pulsed Doppler unit to assess blod flow velocity in scanned vessels. Hemodynamically significant obstruction to renal blood flow is associated with an increased velocity of flow just distal to the obstruction. Early data on the utility of this technique as a screening test are quite positive. However, the quality of the results are highly operator-dependent and confirmation of general utility is needed.

Angiography is the preferred method for establishing the diagnosis and evaluating the anatomy of the renal circulation. Several techniques are available. Intravenous digital subtraction arteriography has several major

disadvantages including poor visualization of the arterial anatomy, motion artefacts and difficulty in interpretation for technical reasons such as obesity or overlying bowel gas (12). Intra-arterial digital subtraction angiography is technically superior to intravenous digital subtraction angiography and results in much better visualization of the renal arteries. Since intra-arterial digital subtraction angiography requires about 15% of the contrast material required for conventional arteriography, uses a smaller catheter and is generally a shorter procedure, it may be the procedure of choice for the diagnostic work-up of ischemic renal disease. At present, it is not in widespread use. Conventional renal arteriography currently is the procedure that best delineates the renal circulation and the presence or absence of collateral blood supply to the kidney. In addition to involving arterial access, it also requires more contrast material than the intra-arterial digital subtraction angiogram. The necessity for angiography to diagnose ischemic renal disease places all patients requiring diagnostic work-up at risk of acute renal failure from the contrast media, as well as the risk of atheroembolic complications from the angiographic manipulation itself.

Although supine and stimulated peripheral renins have been used in the diagnosis of renovascular hypertension, they are unreliable in ischemic renal

disease. This may, in part, be due to the expansion of intravascular volume that commonly occurs in bilateral renal artery stenosis, suppressing plasma renin activity (13). Renal vein renins and split renal function tests are not helpful for the diagnosis of ischemic renal disease since they depend on comparing a response between an affected and unaffected kidney. In the setting of ischemic renal disease, both kidneys are affected and a differential response is frequently not obtainable.

In summary, pending future evaluations of alternate diagnostic maneuvers, such as duplex doppler scanning and magnetic resonance angiography, the diagnosis of ischemic renal disease relies on invasive angiographic demonstration of renal artery stenosis.

MANAGEMENT

The treatment alternatives in ischemic renal disease include conservative medical therapy, percutaneous transluminal angioplasty or surgical revascularization. Medical therapy should only be a choice for those patients who, due to other aspects of their medical condition, are unlikely to tolerate either surgical revascularization or percutaneous transluminal angioplasty procedures.

No prospective studies have yet evaluated the outcome of patients randomized to either surgical versus angioplasty treatment for ischemic renal disease. Numerous case reports and now many published studies have confirmed a relatively high success rate of surgery in preserving, and especially in improving, renal function in patients with ischemic renal disease (14, 15, 16). These studies document that surgical revascularization can result in a dramatic increase in GFR; that it can restore function in the kidney behind a totally occluded main renal artery; and that it can frequently replace nephrectomy for the management of hypertension in renovascular disease with a poorly functioning kidney.

The surgical revascularization procedures which are performed include the aortorenal by-pass, the spleno-, hepato-, ilio-renal by-pass and endarterectomy. Aortorenal by-pass with an autogenous vascular graft is the preferred method of revascularization (23, 24). If however the aorta is seriously diseased, the surgeon may choose to do one of the arterial jump graft procedures. In reviewing multiple surgical series, 60 - 80% of the patients undergoing revascularization have stable or improved renal function. (Table 4)

Percutaneous angioplasty has also been used to reverse renal ischaemia. As can be seen in Table 5, numerous studies have demonstrated that percutaneous transluminal angioplasty can not only be technically successful in opening stenosed arteries, but the initial clinical result is also positive in the majority of patients. In these studies, the lesions were dilated for treatment of renovascular hypertension and a favorable clinical result was an improvement or cure of hypertension. With respect to atherosclerotic lesions, the technical success rate depends on the location and extent of the lesion (17). Atherosclerotic lesions at the origin of the renal artery, ostial lesions, respond poorly to angioplasty with a technical success rate of < 75%. However, in well localized atherosclerotic lesions in the first and second third

of the main renal artery, an initial technical success of 80-95% is achieved by experienced angiographers (Table 5). The re-stenosis rate is 10-30% and the majority of these stenotic vessels can be successfully re-dilated (18). Several recent studies have demonstrated either a return of renal function or long term stability of renal function following dilatation of renal artery stenosis by percutaneous transluminal renal angioplasty (1, 19).

Although there is unequivocal evidence that surgical revascularization or percutaneous transluminal angioplasty can improve and stabilize renal function in patients with ischemic renal disease, there are little data on how many of these patients can avoid dialysis and, importantly, if treatment of the renovascular lesions prolong their survival. The procedures themselves are not without risk. Operative mortality ranges from 2-15%. Operative risk can be predicted by a discriminant analysis of variables which impact on operative mortality. As can be seen in Table 6, the presence of peripheral vascular disease, an abnormal ECG, renal impairment with a serum creatinine > 3 mg/dl, the presence of an aortic aneurysm and the need for a complex procedure all significantly contribute to the operative risk. In the analysis done by Dean (4), a discriminant score of > 10 carries a prohibitive operative risk. Since we know that the majority of patients with atherosclerotic ischemic

renal disease have significant atherosclerotic arterial lesions elsewhere, it is not surprising that their operative mortality or ultimate survival is often poor. Indeed, many authors recommend a careful search for underlying cardiovascular or cerebrovascular disease before proceeding with surgical revascularization of the kidneys. Similarly, percutaneous transluminal angioplasty has a mortality of 1-5% and a risk of significant complications including rupture of the renal artery, dissection of the renal or access artery, renal artery thrombosis, contrast-induced acute renal failure requiring dialysis, or peripheral artery embolization. With these operative and angioplasty risks in mind, the question of the long-term success of these procedures becomes critical. In one uncontrolled study performed by Novick (19), the surgical correction of ischemic renal disease in patients with end-stage renal failure improved survival. Although one must be careful not to extrapolate from this uncontrolled study, the results were sufficiently provocative to suggest that further prospective data should be gathered.

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TABLE 1

CLINICAL PRESENTATIONS OF ISCHEMIC RENAL DISEASE

1. ACUTE RENAL FAILURE (SECONDARY TO REDUCTION IN BLOOD PRESSURE, EMBOLISM OR DISSECTION)

2. PROGRESSIVE AZOTEMIA IN A PATIENT WITH DOCUMENTED RENOVASCULAR HYPERTENSION

3. PROGRESSIVE AZOTEMIA IN A PATIENT WITH REFRACTORY HYPERTENSION (PATIENT AT RISK FOR BUT NOT DOCUMENTED RENOVASCULAR HYPERTENSION)

4. UNEXPLAINED AZOTEMIA IN AN ELDERLY PATIENT

5. HYPERTENSION AND AZOTEMIA IN A RENAL TRANSPLANT PATIENT

TABLE 2

DIAGNOSTIC TESTS FOR IDENTIFYING ISCHEMIC RENAL DISEASE

TEST	COMMENT
1. Rapid-sequence IVP	Not directly helpful. Can be of incidental benefit in identifying patients with one (or one functional) kidney
2. Renal ultrasound	Not directly helpful. Useful to exclude lower urinary tract obstruction. Of incidental benefit to define renal anatomy (1 or 2 kidneys and renal size)
3. Isotopic renal blood flow and functional	Not directly helpful. Can be useful in follow-up to determine patency scans of repaired vessels
4. Angiography	
a) Intravenous digital	May be useful in follow-up after surgery or angioplasty
b) Intra-arterial digital subtraction	May become procedure of choice because of requirement for less contrast media
c) Conventional aortography with selective renal	Currently best test to define renal vascular anatomy
5. Supine and stimulated peripheral renin	Less helpful than in renovascular hypertension. Not primarily indicated
6. Renal-vein renins	Not helpful. Can be useful in deciding which kidney to revascularize first in the setting of bilateral stenosis
7. Split renal function	Not indicated in diagnostic work up. Can be useful in strategy for revascularization
8. Acute hypotensive therapy while monitoring GFR and renal blood flow	Not prospectively useful in diagnosis but occasionally helpful in formulating therapeutic strategy

FROM JACOBSON 1988

TABLE 3

TREATMENT OPINIONS IN ISCHEMIC RENAL DISEASE

1. Conservative Therapy
2. Percutaneous Transluminal Angioplasty
3. Surgical Revascularization
   a. Aortorenal bypass
   b. Spleno-, hepato-, ilio-renal bypass
   c. Endarterectomy

TABLE 4

RESULTS OF SURGICAL REVASCULARIZATION FOR ISCHEMIC RENAL DISEASE

Number of Patients	Mean Age	Operative Deaths	Renal Function			Mean Follow-up	
			Improved Pre-op GFR	Stable	Worse Post-op GFR	Survival	(Mo.)
67 (1975-1980)	60	2%	58%	31%	11%	89%	9
94 (1981-1984)	55						
23	61	4%	65%	21%	14%	78%	24
20	59	15%*	28 ml/min		45 m./min	80%	29
64**	58	***	60%	40%			***

* 3 patients died within 30 days of surgery.

** 58 patients with atherosclerotic disease, 6 patients with fibromuscular dysplasia.

*** Only patients with successful operative procedures were included and long-term follow up was not included.

FROM JACOBSON 1988

TABLE 5

INITIAL TECHNICAL SUCCESS AND CLINICAL RESULTS OF ANGIOPLASTY

STUDY	NO. OF PATIENTS	INITIAL FAVORABLE CLINICAL RESULT TECHNICAL SUCCESS %	ATHEROSCLEROSIS	%FMD
MARTIN 1981	31	83.9	46.2	75
SOS 1982	101	77.2	52.9	87.1
LOHR 1983	128	81.3	NA	NA
TEGTMEYER 1984	109	94.5	93.8	100
MARTIN 1985	100	88	70	85
MILLER 1985	63	87.3	58.8	100
KLINGE 1989	213	80.7	77.4	86.3

Klinge 1989 31 83.9 77.2 94.5

TABLE 6

OPERATIVE RISK VERSUS DISCRIMINANT ANALYSIS

Variable	Discriminant Weight	No. Patients With Variable	Number of Deaths
PVD	2	22	4
+ ECG	2	27	5
Azotemia > 3 mg/dl	4	4	3
AAA	3	29	5
Complex procedure	4	13	4

FROM DEAN 1984

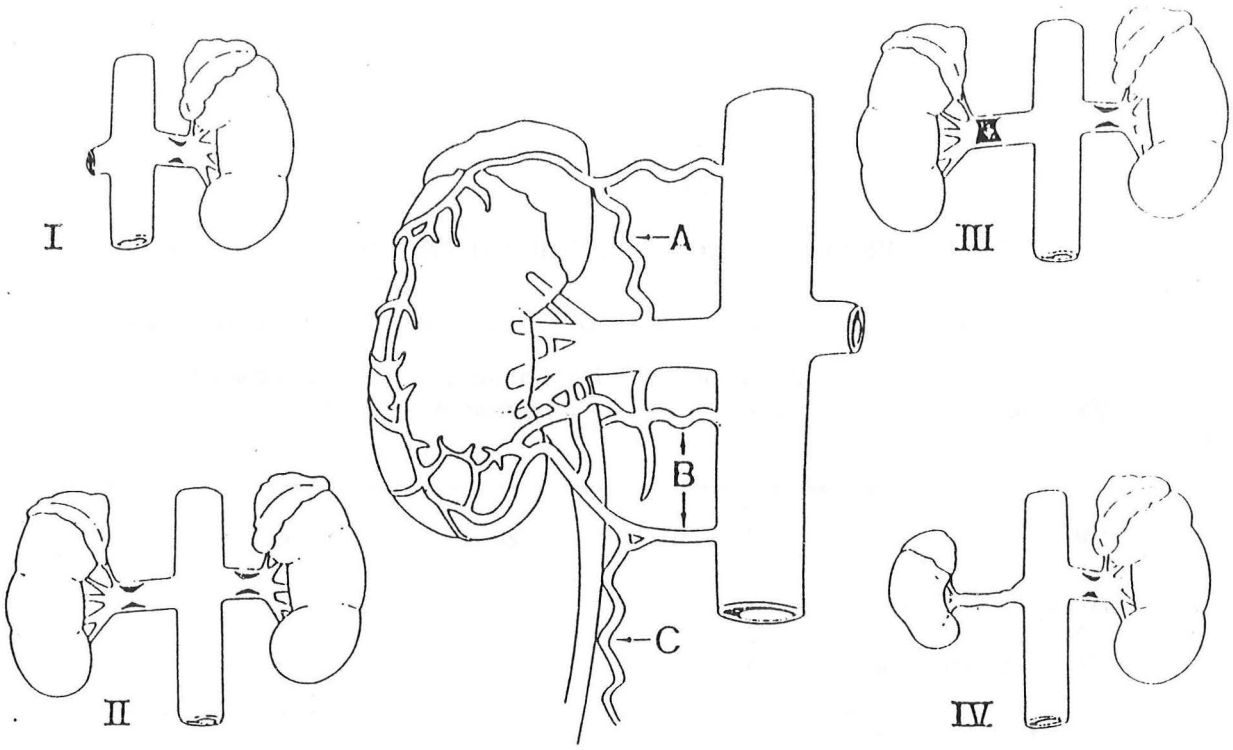

Figure 1. Diagrammatic representation of the collateral circulation which maintains viability of the kidney after occlusion. (see insets). (A) Suprerenal complex (B) Lumbar complex (C) Ureteric complex. The insets illustrate the four most common anatomic findings in atherosclerotic ischemic disease: I. Significant obstruction of an artery to a single kidney; II. Significant bilateral renal artery obstruction; III. Unilateral renal artery occlusion with contralateral significant obstruction; IV. Unilateral significant obstruction with a poorly functioning (i.e. congenitally hypoplastic, end stage, reflux, etc.) contralateral kidney.