

Reversible dialysis-dependent renal failure due to undiagnosed renovascular disease

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ABSTRACT

Renovascular disease (RVD) can present with resistant hypertension, acute or rapidly progressive renal failure and occasionally nephrotic proteinuria. Revascularization plays an important role in controlling blood pressure and preserving renal function. It is widely believed that delay in revascularization would result in irreversible loss of renal function. However, we report a favorable outcome despite delayed revascularization in two patients of RVD- one presenting with recurrent flash pulmonary edema and other with progressive renal failure. The former's serum creatinine returned to normal despite 3 months of anuria and the latter became dialysis-independent despite 2 months of progressive decline in renal function. Both remain dialysis-free 3 years after surgery.

Key words: Atherosclerosis, renovascular disease, surgical revascularization

Introduction

Renovascular disease (RVD) is a potentially reversible cause of hypertension and renal failure. It presents with a wide spectrum of clinical manifestations ranging from difficult-to-control hypertension, flash pulmonary edema, progressive renal failure, nephrotic proteinuria to incidental detection on angiography.^[1] Although evidence for routine renal artery intervention in patients with ischemic nephropathy is not bolstering, some authors recommend intervention in advanced chronic kidney disease (CKD)^[2] and even for asymptomatic renal artery lesions to prevent inevitable and irretrievable loss of renal function.^[3,4] We report the cases of two young males presenting with dialysis dependent renal failure who underwent delayed surgical revascularization with a gratifying outcome.

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Access this article online	
Quick Response Code:	Website: www.indianjnephrol.org
	DOI: 10.4103/0971-4065.101267

Case Reports

Case 1

A 42-year-old male, active smoker and a known hypertensive for 6 years on treatment with amlodipine 5 mg and atenolol 50 mg, presented with headache and exertional breathlessness of 1-month duration. Evaluation revealed pallor, pedal edema, absent pulses in lower limbs, blood pressure 230/130 mmHg in both upper limbs, Keith Wagner Grade III hypertensive retinopathy (AV ratio 1 : 3, hard exudates and flame-shaped hemorrhages), bruit over right renal, epigastric area and no carotid bruit. Cardiac examination revealed cardiomegaly with no gallop. Diagnosis of RVH was considered and investigated. Labs showed Hb 10.4 gm%, Blood urea – 230 mg/dl, serum Creatinine – 23.8 mg/dl and serum K⁺ 4.3 mEq/L. Ultrasound showed shrunken right kidney with normal sized left kidney (9.2 × 4.2 cm) and 2D echo showed left ventricular hypertrophy.

His blood pressure remained poorly controlled despite four antihypertensives (clonidine 0.4 mg, nifedipine 60 mg, atenolol 100 mg, prazosin 10 mg). Hemodialysis was initiated. Renal angiography done after initial stabilization revealed occlusion of aorta just below the origin of superior mesenteric artery, nonvisualization of both renal arteries and extensive collaterals in the infrarenal area [Figure 1]. A delayed left nephrogram was seen on screening after 2 hours. After 3 months of anuria and recurrent attacks of flash pulmonary edema, the initially reluctant patient agreed

for revascularization surgery. The renal artery was found to be patent just proximal to its bifurcation and an end-to-side spleno-renal bypass was performed [Figure 2]. The kidney showed immediate diuresis and no further dialysis was required. His serum creatinine dropped from 12.2 to 2.2mg/dl at 1 week and 1.2 at 2 months and has remained stable for the last 3 years. He is normotensive on three drugs: enalapril 10mg, clonidine 0.2mg and nifedipine 30mg in divided doses. Histopathological examination of renal artery biopsy showed atherosclerotic changes.

Case 2

Another 52-year-old male, a chronic smoker with poorly controlled hypertension of 5 years presented with malaise and anorexia since 2 months. Patient was diagnosed as rapidly progressive renal failure with accelerated hypertension and initiated on hemodialysis. His weight was 48 kg with pallor, no edema; BP-160/100mmHg, all peripheral pulses palpable; systemic

exam was unremarkable. Labs showed Hb – 9.8gm%; TLC- 11,200/mm³, urinalysis showed 3+ albuminuria with protein creatinine ratio 2.1, urea - 166mg/dl; S-creatinine: 6.3mg/dl; S K+ 4.2 mEq/L. Ultrasound abdomen showed small right kidney and normal-sized left kidney. MRI abdomen showed diffuse atherosclerotic aorta, bilateral proximal renal artery stenosis and focal saccular aneurysm of right renal artery [Figure 3]. Two-dimensional echo showed normal left ventricular function with minimal pericardial effusion. Conventional angiogram showed nonvisualization of right renal artery and >90% stenosis on the left. Percutaneous transluminal renal angioplasty (PTRA) was done [Figure 4] with good result. Transient increase in urine output (500-700 ml/day) was noted but he reverted back to oliguria after 5 days due to stent thrombosis which was confirmed on CT angiogram. Hemodialysis was continued. His BP control was poor despite five classes of antihypertensive drugs. Radionuclide isotope renography (DTPA isotope) showed good tracer uptake by left kidney suggesting its viability. Renal artery bypass (left common iliac to left renal artery with reverse saphenous venous graft) was done subsequently and brisk diuresis was noted postoperatively with a urine



Figure 1: Renal angiogram showing occlusion of aorta just below the origin of superior mesenteric artery, with nonvisualization of both renal arteries and extensive collaterals in the infrarenal area

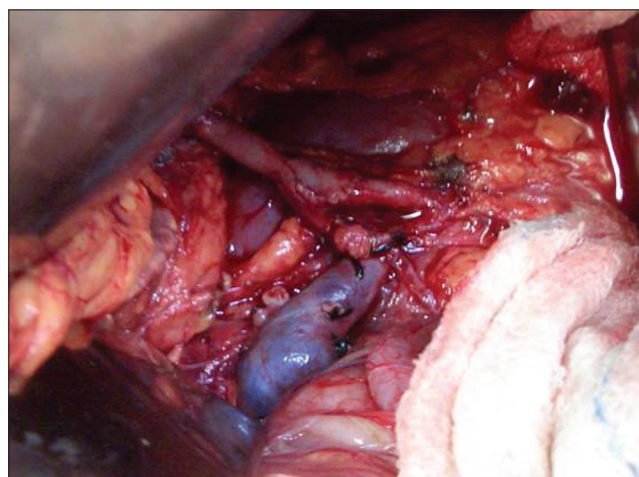


Figure 2: Spleno-renal bypass surgery

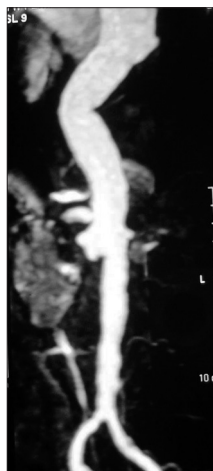


Figure 3: Diffuse atherosclerotic aorta with bilateral proximal renal artery stenosis and focal saccular aneurysm of right renal artery

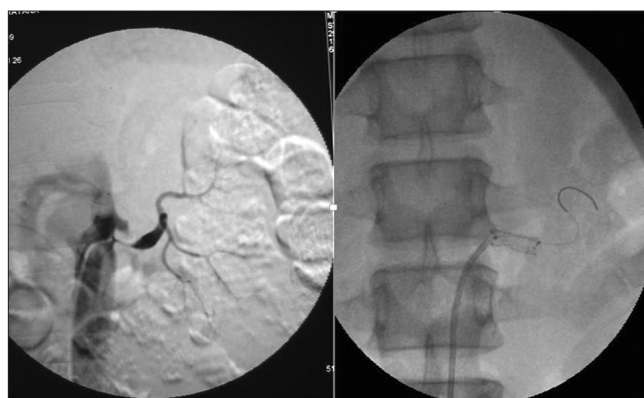


Figure 4: Percutaneous transluminal renal angioplasty with stent to left renal artery

output >3L/day. He required only two more hemodialysis postsurgeries and was discharged with S-creatinine – 3 mg/dl, BP110/80 (on three classes of antihypertensives) after 2 months of continued intermittent dialysis. Follow-up at 3 years showed his S-creatinine to be 4mg/dl without any need for dialysis.

Discussion

The goal of management of any form of RVD is control of blood pressure, preservation of renal function and avoidance of complications of treatment. However, the choice between three potential mode of treatment namely medical, surgical and PTRAs is without any consensus because of various uncertainties regarding the likely outcome in a patient having diffuse atherosclerosis.

The role of renal revascularization in RVD remains controversial^[2-5] especially due to operative mortality of over 7% and progression to dialysis dependence in nearly one-third of patients over 3 years despite intervention.^[6] Even the data regarding the mortality and dialysis risk associated with untreated atherosclerotic renal artery disease with or without renal insufficiency is inadequate further compounding the decision making. One study showed a 4-year estimated survival of 65% in patients with unilateral renal artery stenosis of >50% - a significant reduction in comparison to age and sex-matched controls without renal artery stenosis.^[7] Another study showed a 40% mortality within 2 years and progression to dialysis in 35% over 5 years in patients with bilateral renal artery stenosis who were medically managed.^[8] It has been suggested that small kidney size (<8 cm), marked chronic azotemia (>3.5 mg/dl), poor retrograde filling of collaterals, high resistance index (>80) of renal artery, failure to visualize kidney on isotope renography and sclerosed glomeruli on histology are markers for lack of salvagability. Several reports suggest poor outcome with intervention in patients with renal failure in terms of blood pressure control, renal function preservation and survival with higher incidence of complications. The ASTRAL trial depicted that there is no significant difference between revascularization and medical therapy when taking in to account the rate of progression of renal impairment and the rates of new onset kidney injury, initiation of dialysis, renal transplantation, nephrectomy or death from renal failure and major cardiovascular events.^[9] One recent study showed that renal artery revascularization even resulted in improved control of heart failure and a reduction in hospitalizations secondary to it.^[10] These conflicting facts should not decrease the enthusiasm for intervention because such patients have greatest potential for absolute benefit from renal as well as cardiac point of view.^[10,11] Therefore, rather than

absolute renal dysfunction, the rapidity of decline in renal function preceding surgery and severe occlusive disease (with collaterals) affecting entire renal mass, even with increased serum creatinine level is associated with high probability of improved postoperative renal function as seen in our patient.

Occasionally, renal biopsy may be helpful to determine the suitability for renal revascularization (lack of ischemic obsolescence of glomeruli and tubules suggesting viability). In both of our patients, though the size of the left kidney was normal with faint delayed nephrogram on angiogram, kidney biopsy was not felt safe because of uncontrolled hypertension and dialysis dependency. Although it is believed that longer duration of renal artery occlusion may result in irreversible injury, there are reports of improvement in renal function despite prolonged period of anuria as in our case. In one report of 9 dialysis dependent end-stage renal disease (ESRD) patients of 1 week to 13 months due to atherosclerotic RAS who underwent surgical revascularization immediate recovery of renal function postoperatively was noted in all, and none required further dialysis.^[11] In another report of nine ESRD patients of atherosclerotic RAS etiology who underwent surgery, six of them had extended survival with freedom from chronic dialysis.^[12] Our patient recovered renal function after surgical revascularization with no dialysis requirement in follow-up of 3 years. Still others^[13] have demonstrated the potential of recovery of renal function irrespective of kidney size, angiographic evidence of collateralization or reconstitution of the distal renal artery. This encouraged us to explore the left renal artery though we failed to demonstrate it on angiography in first patient.

Degree of renal dysfunction alone should not be the criteria to deny definite surgical option. In selected patients with severe hypertension and ischemic nephropathy, renal artery revascularization can retrieve significant renal function. Early renal function response has a bearing on time to progression to dialysis and on dialysis-free survival. The method of revascularization should be selected with the aim of retrieving maximum renal function.

These two cases highlight the salutary benefits of appropriate surgical intervention. The presence of collaterals (near normal kidney size and echo-texture) could be taken as a potential clue of viability of ischemic kidney. There is no room for pessimism based on projected negative factors like anuria, marked azotemia, dialysis dependency and each case merits focused assessment of individual risk benefit analysis thereby avoiding hasty decisions.

Acknowledgment

We thank our colleagues and staff of Nephrology and Vascular surgery.

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How to cite this article: Jha R, Gude D, Narayan G, Mandal SN, Gupta PC. Reversible dialysis-dependent renal failure due to undiagnosed renovascular disease. *Indian J Nephrol* 2012;22:314-7.

Source of Support: Nil, **Conflict of Interest:** None declared.