INTERVENTIONAL RADIOTHERAPY in Renal Disorder

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INTRODUCTION

The field of interventional radiology has developed rapidly and is used by physicians in all fields of medicine. With the development of better computer-assisted imaging of internal organs and developments of less traumatic thin profile guide wires, balloons, stents, etc. has led to more and more physicians taking up various interventional procedures which demand high technical skill. These procedures lead to more accurate diagnosis and avoid major surgery, often in high risk patients. A successful and safe outcome is specific to the clinician performing the procedure itself and to intra and post procedure care by the team. Most of these procedures have a steep learning curve. Interventional radiology procedures in renal disorders are shown in Table 1.

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<th>Interventional Radiology In Renal Disorders</th>
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<td>A. Arterial</td>
<td>1. Angiography /plasty + stent</td>
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<td>2. Angio-infarction</td>
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<td>3. Fistulogram [for Malfunctioning of A V Fistula for dialysis]</td>
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<td>1. Central venous cannulation for temporary vascular access for haemodialysis</td>
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<td>A. Noncuffed catheters</td>
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<td></td>
<td>B. Cuffed catheters</td>
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<td></td>
<td>2. Venography</td>
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Note: Arterial interventions may be done for suspected native kidney or transplanted kidney artery disease.

2. Kidney biopsy

- Native kidney
- Transplanted kidney

VASCULAR INTERVENTION

A. Renal angiography and angioplasty

The commonest indication for this is renovascular disease leading to renovascular hypertension or ischaemic nephropathy or both. In selected group of hypertensive patients specially those having renal insufficiency without primary renal parenchymal disease; renovascular disease may account for as many as 40% of cases. Renovascular disease fortunately is a correctable cause of hypertension in vast majority of patients. It must however be remembered that renovascular disease may coexist with essential hypertension and significant stenosis could be found among normotensive individuals. It may affect native kidneys or the renal graft artery and may or may not lead to clinically obvious deterioration of renal function. It must also be noted that renovascular disease may affect unilaterally or bilaterally and may affect major branches of the renal artery. The renal artery may be involved in isolation or in association with vascular tree elsewhere. The diseases of renal artery manifests as renal artery stenosis, renal artery aneurysm or thrombosis. The common diseases affecting renal artery in order of commonness in our country are: 1. Atherosclerosis 2. Aortoarteritis 3. Fibromuscular dysplasia [Fig.1]. Atherosclerosis constitutes the predominant cause accounting for 70 to 85% of our cases.

Considering a very large number of hypertensives; it is not possible to screen every hypertensive for renovascular disease. Therefore, patients have to be selected for investigation of renovascular disease based on strong clinical suspicion and initial investigations. The commonest reasons, which lead to strong suspicion of renovascular disease are shown in Table 2. In our study of 140 patients who underwent renal angiography based on clinical suspicion the relative risk of predicting renovascular disease is shown in Table 3.1

Renovascular hypertension is said to be present when on treatment of the anatomical renal artery le-
sion leads to cure of hypertension and hypertension recurs or restenosis. Ischaemic nephropathy is defined as clinically important reduction in glomerular filtration rate or loss of renal parenchyma caused by haemodynamically significant renal artery stenosis. Atherosclerotic renovascular disease and ischaemic nephropathy are well recognized; however, they are very under-diagnosed due to lack of strong index of suspicion among physicians and perceived invasive nature of investigations.

**Incidence**

It is difficult to know the exact incidence of renovascular disease among hypertensives. Various series have reported the prevalence to vary between 0.2% and 32% (Olin C.W.1998). In carefully selected group of hypertensives with a strong clinical suspicion the prevalence may be in the range of 45 to 60%.

Liu et al. 1997 in a 12 year autopsy study reported on prevalence and predictors of renal artery stenosis. Out of 297 patients with evidence of myocardial infarction atherosclerotic renal stenosis (ARAS) was found in 35 (12%) and 10 had bilateral disease. In patients with hypertension proteinuria and renal insufficiency renal artery stenosis was seen in 19%, 39% and 39% respectively. These were identified as independent predictors of risk of renal artery stenosis by multiple regression analysis; the risk increased by 3.4, 13.5 and 4.8 fold in patients with myocardial infarction.

In another series 22 out of 76 (29%) patients having coronary artery disease, had more than 50% renal artery stenosis (Ventrovac 1989). Similarly (Harling et al. 1992) reported 164 (20%) out of 817 had significant renal artery stenosis when screened during coronary angiography.

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<th>Table 2</th>
<th>Reasons For Suspecting RVHT</th>
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<td>1.</td>
<td>HT diagnosed before age 40 years</td>
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<td>2.</td>
<td>Newy diagnosed HT after age 55 years</td>
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<td>3.</td>
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<td>5.</td>
<td>Unilateral small kidney</td>
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<td>6.</td>
<td>Evidence of peripheral vascular disease / coronary artery disease</td>
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<td>7.</td>
<td>Hyperkalaemia, metabolic alkalosis before treatment</td>
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<td>8.</td>
<td>Presence of renal bruit</td>
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<td>9.</td>
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<th>Table 3</th>
<th>Relative risk of clinical causes for suspecting RVHT: Study of 140 patients of renal artery disease at MPUH, Nadiad</th>
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<tr>
<td>Causes</td>
<td>R. R.</td>
</tr>
<tr>
<td>1. Newly diagnosed HT after age 55 yrs.</td>
<td>0.01</td>
</tr>
<tr>
<td>2. Worsening HT after age 55 yrs.</td>
<td>0.35</td>
</tr>
<tr>
<td>3. HT Retinopathy grade III and IV without renal-parenchymal disease</td>
<td>0.91</td>
</tr>
<tr>
<td>4. Unilateral small kidney</td>
<td>0.91</td>
</tr>
<tr>
<td>5. Evidence of peripheral vascular disease</td>
<td>0.31</td>
</tr>
<tr>
<td>6. Hyperkalaemia metabolic alkalosis</td>
<td>0.03</td>
</tr>
<tr>
<td>7. Presence of renal bruit</td>
<td>0.91</td>
</tr>
<tr>
<td>8. Rise in serum creatinine after ACEI</td>
<td>0.91</td>
</tr>
<tr>
<td>9. Recurrent pulmonary oedema</td>
<td>0.03</td>
</tr>
</tbody>
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R. R. Relative Risk (Rajapurkar, 1995 W USI, Goa) 2  
Spec. Specificity  
Sen. Sensitivity  
PPV Positive Predictive Value  
NPV Negative Predictive Value  

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In a recent study of 395 consecutive patients with peripheral vascular disease who had no clinical clue to suspect renovascular disease; 38% with abdominal aortic aneurysm, 33% with aorto-iliac disease and 39% with lower extremity occlusive disease also had renal artery stenosis [Olin J.W.1998].

In a prospective study including 149 patients with suspected renovascular disease, severe ARAS, carotid artery disease and peripheral arterial disease was seen in 44%, 19% and 21% respectively and severity of the renal artery disease increased with increasing degree of other arterial disease. Prevalence of severe carotid artery disease increased from 7% in mild renal artery disease group to 28% in the severe renal artery group [Beach et al 1998].

Thus patients having atherosclerosis elsewhere specially those having coronary artery disease, abdominal aortic aneurysm, peripheral vascular disease or carotid artery disease have high prevalence of significant renal artery disease; even in the absence of usual clinical clues to suspect renal artery stenosis. Diabetics have been shown to have prevalence similar to nondiabetics [Olin J.W. 1990].

**Natural History**

Like lesions of atherosclerosis elsewhere the ARAS is progressive disease. This leads to progressive narrowing and ultimately to occlusion leading to loss of the kidney. Clinically this manifests as progressive worsening of hypertension, renal atrophy and loss of GFR. It is not possible to predict clinically which lesions will progress. Progression may also be clinically silent.

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**Fig.1**

Results of Renal angioplasty at Our institute.
Table 4
Showing results of angioplasty without stent for atherosclerotic renal artery disease at MPUH, Nadiad-1998

<table>
<thead>
<tr>
<th></th>
<th>A Crt&lt;1.6 [n=16]</th>
<th>B Crt 1.7-2.9 [n=9]</th>
<th>C Crt. 3-6.9 [n=18]</th>
<th>D Crt. &gt; 7 [n=10]</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>SP control Achieved</td>
<td>14 [88%]</td>
<td>4 [45%]</td>
<td>7 [39%]</td>
<td>9 [90%]</td>
<td>35/53 [66.03%]</td>
</tr>
<tr>
<td>BP Before After</td>
<td>188/112 146/87</td>
<td>207/111 153/89</td>
<td>178/105 153/89</td>
<td>192 148/81</td>
<td>-</td>
</tr>
<tr>
<td>Anti-HT Before After</td>
<td>2.6 1.1</td>
<td>2.9 1.5</td>
<td>2.8 1.7</td>
<td>3.1 1.8</td>
<td>-</td>
</tr>
<tr>
<td>Reduction in Creatinine</td>
<td>No Deterioration</td>
<td>4 [45%] [&gt;0.5]</td>
<td>7 [39%] [&gt;1]</td>
<td>4 [40%] Off dialysis</td>
<td>15/37 [40.54%]</td>
</tr>
</tbody>
</table>

Primary technical failure 5 out of 73 vessels [6.84%] [Rajapurkar, 1999]15

Table 5
Indications for renal biopsy at MPUH, Nadiad

A. Glomerular disease

1. All adults suspected of glomerular disease excluding
   a. Longstanding diabetics with nephrotic syndrome and retinopathy
   b. Nephrotic syndrome of pregnancy
   c. Nephrotic syndrome in patients with advanced malignancy.
2. Children with glomerular disease excluding:
   a. Nephrotic syndrome; having no renal failure, no urinary sediments which are steroid-responsive.
   b. Clinically typical post infectious glomerulonephritis which resolves within 8 weeks of onset.

B. Renal failure

All patients with renal failure for which cause, reversibility and duration cannot be ascertained by clinical examination and other laboratory tests.

C. Transplanted kidney biopsy

a. Graft dysfunction
b. Recurrence of original disease

Table 6
Relative contraindications for renal biopsy at MPUH, Nadiad

- Single kidney status, specially if it is abnormally located
- Polycystic disease.*
- Severe bleeding/coagulation abnormality
- Obstructive uropathy with gross thinning of cortex.*

* If very strongly indicated we have to perform open surgical renal biopsy in these situations.

years and 48% at 3 years. 4 renal arteries progressed to occlusion all had > 60% stenosis at initial assessment.

Strandness 19949 reported that kidneys having > 60% renal artery stenosis 26% had decrease in renal length by more than 1 cm over average follow up period of 14.4 months. The estimated risk of loss of more than 1 cm length was 19% at 1 year.

Atherosclerotic renovascular disease is probably a frequent cause of end stage renal disease among elderly patients on dialysis. Coen et al10 in a prospective study of 133 hypertensive chronic renal failure patients above the age of 50 years; found haemodynamically significant stenosis in 3.2% in the 50 to 59 years age group, 20% in 60 to 69 years age group and 25% in more than 70 years age group.

Diagnosis

No clinical algorithm has been developed that has significant accuracy to provide a reliable diagnosis of renovascular hypertension from clinical features.
Commonly employed preliminary noninvasive tests are duplex or colour Doppler scanning; Captopril radioisotope scanning; magnetic resonance angiography or CT angiography. These are done in addition to clinical examination; renal function tests, urinalysis and serum electrolytes and bicarbonate estimation. Most of these tests are observer and equipment dependent involving a learning curve. Their sensitivity, specificity, positive and negative predictive values have been therefore reported over a wide range in literature. Each institution should therefore evolve a system of assessing clinically suspected renal artery disease based on availability of experienced personnel and quality of equipment before subjecting patient to angiography.

Physiological predictors of outcome and for lateralising following tests are most useful: a. differential renal vein renin estimation and b. Captopril renogram.

**Management**

There is enough evidence to suggest that renal revascularisation; either surgically or percutaneously, leads to: a. better blood pressure control, b. improvement of renal function c. preservation of renal function, d. slower decline of GFR, e. reduction of need for antihypertensive medication and in few dialysis dependant patients f. avoids need for dialysis.

Following revascularisation procedures are available: a. percutaneous transluminal renal angioplasty with or without endovascular stenting of the stenotic lesion and b. surgical revascularisation.

Both procedures have significant procedure related morbidity and mortality. Widely varying results reported in literature probably reflect the initial selection of patients, comorbid conditions at the time of revascularisation and the centre’s experience.

Klow et al 1998 reported 92% initial angiographic success in 295 patients with ARAS. 8% hypertensives were cured and 58% improved. 2 deaths and 4.7% clinically significant complications were seen in this series.

Von Knorring et al 1996 in a 4 year follow up of 38 ARAS reported 92% primary success rate. 4 patients developed restenosis, 12 of these patients had bilateral disease. 85% patients had long term benefit, 11% were cured, 74% improved and 15% were failures. 75% with bilateral ostial disease had long term benefits. 2 patients died of acute myocardial infarction.

Fiala et al, 1998 reported 95% immediate technical success rate in 21 patients with bilateral athero-
sclerotic renal artery ostial lesions. The mean arterial blood pressure improved and serum creatinine decreased significantly over a mean follow up of 2 years. Renal angioplasty with primary stenting was done. The cumulative restenosis rate was 18% at 2 years. Major complications occurred in 4 patients, no mortality was seen.

In our initial study of 53 patients having ARAS who underwent angioplasty were reviewed. 33 patients had unilateral involvement and 20 had bilateral renovascular disease. There were 11 ostial lesions included. 79 percutaneous transluminal renal angioplasty (PTRA) procedures were carried out on 73 vessels no stents were placed. Six patients had ostial lesions. The study included 42 males and 11 females. The average age was 49 years. 19 diabetics were included the mean follow up was 14 months. The patients were classified in 4 groups according to their serum creatinine. The patients were analyzed for improvement in blood pressure control, reduction in anti-hypertensive drug requirement, reduction in serum creatinine and becoming dialysis independent. The results are shown in Table 4.15

Subsequent to this study we have done angioplasty and stent placement in 153 patients on a total of 172 arteries. Out of these 153 patients 75.5% were male and 23.5% were female. Their mean age was 47.2±16.0 years. 92.2% had atherosclerotic disease, 7.8% had aortoarteritis. 39.2% had bilateral renal artery disease. In this series 88.7% patients serum creatinine either stabilized or improved. Our primary technical success rate was 94.1%. There was significant reduction of antihypertensive medicines needed for these patients. 84% had better blood pressure control at 1 year after the procedure. We had two procedure related deaths and minor complications were 7.9%. We have an incidence of 12.4% contrast nephropathy. All patients of contrast nephropathy recovered renal function to pre-procedure level.

B. Angio-infarction

The commonest indication for angio-infarction is life-threatening haemorrhage following trauma to the kidney. Commonest cause of trauma to the intra-renal vessels is during kidney biopsy or percutaneous surgery for stone disease. The trauma leads to arterio-venous fistula/malformation and bleeding. The bleeding vessel is identified by doing selective angiogram. Various embolising material like coils, gelfoam etc. could be used to block the artery supplying the A V Fistula / malformation which is bleeding. This leads to arrest of bleeding and hence is life saving. [See fig.2].

Other indications for angio-infarction of the kidney could be to block the main renal artery prior to nephrectomy for large vascular renal tumour.

C. Venous Interventions

Central venous cannulation for temporary vascular access for haemodialysis: Haemodialysis is used as replacement for kidney function in patients of acute kidney failure and chronic kidney failure. Haemodialysis requires 300 to 400 ml of patients blood to be removed and similar amount returned to the patient every minute after passing through the dialyzer. This amount of flow is possible only in arteries or central veins through double lumen catheters. These catheters may be cuffed or noncuffed. These are inserted preferably into the internal jugular, femoral or subclavian vein under fluoroscopy guidance. The veins are initially punctured 'blindly' or by using doppler ultrasound to localise the vein. The catheter is then threaded over a guide wire passed through the puncture needle and the wire is removed leaving the catheter in situ.

Kidney Biopsy

Needle biopsies of kidneys have revolutionized understanding of the evolution of disease process apart from their pathology/pathogenesis much before irreversible loss of function leading to end stage failure has occurred. With advances in imaging techniques; such as real time ultrasound/ CT guidance and better needles and spring loaded automated devices, there has been marked improvement in diagnostic yield as well as safety of biopsy procedure, in adequately trained hands.

INDICATIONS FOR RENAL BIOPSY

The indications for doing a renal biopsy vary from centre to centre and from individual to individual. Indications and contraindication for renal biopsy at our institution are outlined in Tables 5 and 6. Rarely there are other uncommon situations where renal biopsy will help e.g.Oxalosis, Alport's disease, etc.

The commonest reason for doing kidney biopsy of transplanted kidney is to make definitive diagnosis of a cause of graft dysfunction. The causes of graft dysfunction could be a. rejection, acute or chronic; b. acute tubular necrosis or c. Cyclosporin toxicity.

The technique of renal biopsy has been refined over past four decades. With the advent of real time ultrasonography, localisation of the kidney became much easier and biopsy yield improved and complications declined. The latest technological advances are the spring loaded semiautomatic biopsy devices.
which make the procedure much simpler and probably reduce the post biopsy complications.

At our center we use both manual as well as semi-automatic, spring loaded devices under real time ultrasound localization using 14 or 16G “Trucut” needle depending on the choice of the nephrologist.

The indications for renal biopsy in nephrotic syndrome have been widely debated and there are a few authorities who have recommended empirical steroids [without a renal biopsy] in patients with nephrotic syndrome. However, we and many others believe that the renal biopsy in experienced hands carries less risk than an empirical trial of steroids. Dr.M.K.Mani from the Apollo Hospital, Chennai in a study to compare the risk of renal biopsy versus empirical steroid therapy has shown that the risk of such therapy is 12 times greater than the risk of biopsy. It is our policy therefore, to explain the risks and benefits of biopsy to the patients before doing the procedure.

Renal biopsy has evolved over the decades into a procedure, which in a well equipped centre carries minimal risk and can give a lot of information. The latter is very useful for planning therapy and disease prognostication.

SUMMARY

Like in other field of medicine interventional radiology procedures have immensely contributed to correct diagnosis and therapy of patients with renal disorders. Renovascular disorders are not uncommon and results of interventional procedures in good hands are gratifying. Renal biopsy is an essential tool for correct diagnosis and hence treatment of all glomerular disease and also for correct management of dysfunction of transplanted kidney.

REFERENCES


