BMJ Best Practice

Renal artery stenosis

The right clinical information, right where it's needed

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Summary

- Typically due to atherosclerotic disease or fibromuscular dysplasia.
- Often presents with accelerated or difficult-to-control hypertension.
- Worsening kidney function, especially after initiating renin-angiotensin blockade, and recurrent flash pulmonary oedema are common features.
- Presence of renal artery narrowing does not necessarily indicate clinical consequences. Renal artery stenosis, renovascular hypertension, and ischaemic nephropathy are various manifestations of this process.
- Definitive diagnosis is with imaging.

BASICS

Definition

Renal artery stenosis (RAS) is a narrowing of the renal artery lumen. It is considered angiographically significant if more than a 50% reduction in vessel diameter is present.[1] Ischaemic nephropathy is a chronic reduction in glomerular filtration rate that occurs from a narrowing in the renal artery. Renovascular hypertension is hypertension mediated by high levels of renin and angiotensin II, produced by an underperfused kidney supplied by a stenosed renal artery.

Epidemiology

RAS has a prevalence of 0.2% to 5% in all hypertensive patients.[7]

Epidemiology depends on the underlying cause:

- Atherosclerotic RAS accounts for 90% of all RAS.[1] [8] [9] Prevalence is as high as 25% in patients with CAD undergoing cardiac catheterisation.[2] Two percent of end-stage renal disease (ESRD) is due to ischaemic nephropathy.[10]
  [Fig-1]
- Fibromuscular dysplasia accounts for 10% of clinical RAS.[11] Females are 2 to 10 times more likely than males to be diagnosed with this form of RAS.[2] [11] Onset typically occurs before the age of 30.[2] [11]
  [Fig-2]

Aetiology

Atherosclerotic RAS[2] [12]
[Fig-1]

- Atherosclerosis
- Diabetes mellitus
- Dyslipidaemia
- Smoking.

Fibromuscular dysplasia
[Fig-2]

- Medial fibroplasia (histological finding in 90% of cases)[2] [11]
- Intimal and adventitial fibroplasia (less common)[2] [11]
- Smoking.[11]

Other causes of renal artery disease[1] [12]

- Post-transplant (site of vascular anastomosis)
- Miscellaneous renal arterial disease
- Renal artery aneurysm
- Accessory renal artery
- Takayasu's arteritis
- Atheroemboli
- Thromboemboli

- Williams syndrome
- Neurofibromatosis
- Spontaneous renal artery dissection
- Arteriovenous malformations
- Arteriovenous fistulas
- Trauma
- Abdominal radiotherapy
- Retroperitoneal fibrosis.

Pathophysiology

In general:[1] [2] [3] [7]

- Activation of the renin-angiotensin system causes increased systemic vascular resistance and sodium retention.
- When the stenosis exceeds 50% reduction in vessel diameter, these regulatory mechanisms may fail, leading to worsening kidney function and difficult-to-control hypertension.
- Underperfusion of the kidney caused by blood flow obstruction produces adaptive changes in the kidney, including atrophy of tubular cells, fibrosis of the capillary tuft, and intra-renal arterial medial thickening.
- Angiotensin II stimulates fibroblast activity, which may lead to fibrosis in the glomerular tuft and in the tubules.
- In addition to activation of the renin-angiotensin system, other mechanisms include activation of the sympathetic nervous system, abnormalities in endothelial nitric oxide, endothelin release, and increased oxidative stress.[3]
- Hypertension can cause hyalinosis, mesangial cell expansion, and growth factor release resulting in fibrosis.
- Bilateral RAS results in volume overload with inappropriately elevated levels of renin.

Atherosclerotic RAS:[1] [2]

- Usually involves the ostial and proximal third of the renal artery.
- Endothelial injury and atherogenesis.
- Spontaneous or iatrogenic atheroemboli may further deteriorate kidney function.

Fibromuscular dysplasia:[1] [11]

- Typically involves the distal two-thirds of the main renal artery, as well as secondary and tertiary branches.
- Unknown aetiology.

Classification

Anatomical[2] [3] [4] [5]

- Bilateral
- Unilateral
- Unilateral in a single functional kidney

BASICS

- Proximal
- Distal

Severity[6]

- Moderate stenosis: 50% to 70% reduction in vessel diameter
- Severe stenosis: >70% reduction in vessel diameter
- Total occlusion: 100% reduction in vessel diameter

Primary prevention

No studies have been done to evaluate primary prevention of atherosclerotic RAS. A reasonable approach would suggest that aggressive cardiovascular risk factor modification may be beneficial.

Secondary prevention

- Patients should be referred to a nutrition specialist for evaluation and counselling regarding a low-salt, low-cholesterol diet. Special populations may require more specific advice (i.e., patients with diabetes or chronic kidney disease).
- Consideration should be given to enrolment in an exercise programme. If this is not possible, patients should be encouraged to exercise for half an hour every day.
- Smoking cessation or non-initiation is advised.

Case history

Case history #1

A 68-year-old man with known coronary artery disease and peripheral vascular disease presents with recurrent episodes of flash pulmonary oedema, worsening kidney function, and progressively difficult-to-control hypertension. An angiogram of the aorta and renal arteries shows a sclerotic aorta with plaque extending into the proximal third of both renal arteries.
[Fig-1]

Case history #2

A 32-year-old woman with no prior medical history is seen for worsening headache and is found to have a BP of 180/110 mmHg. Her BP responds inadequately to thiazide diuretics and calcium-channel blockers. A magnetic resonance angiogram of the renal arteries reveals a beaded appearance indicative of fibromuscular dysplasia.
[Fig-2]

Other presentations

RAS due to fibromuscular dysplasia is more common in women <30 years. RAS in general often presents as accelerated, resistant, or malignant HTN. RAS may be associated with acute decline in kidney function after initiation of renin-angiotensin blockade. It may be diagnosed in assessing an unexplained atrophic kidney, or discrepancy in kidney size >1.5 cm, or sudden, unexplained, and/or recurrent pulmonary oedema.

DIAGNOSIS

Step-by-step diagnostic approach

The evaluation of RAS includes consideration of factors in the patient's history and decisions on the appropriate imaging modalities.

History

Age of onset of hypertension (HTN) may be suggestive of the underlying aetiology of RAS:

- <30 years suggests fibromuscular dysplasia (FMD).[7] [14]
- >55 years suggests atherosclerotic RAS.[7] [14]

Sudden or unexplained recurrent pulmonary oedema is suggestive of RAS:[7] [14] [15] [16]

- In patients with atherosclerosis, RAS may induce an acute or subacute acceleration of a pre-existing essential hypertension that may precipitate flash pulmonary oedema.

Hypertension (accelerated, malignant, or resistant)

- Patients with either atherosclerotic or FMD form of RAS can present with severe, progressive, and/or difficult-to-control HTN, sometimes causing end-organ damage.[7] [14]

Kidney dysfunction or acute kidney injury:

- Unexplained kidney dysfunction may result from progressive stenosis or HTN-related end-organ damage.[2] [7]
- Acute kidney injury can be seen in some patients with bilateral RAS or RAS of a single functioning kidney after starting an ACE inhibitor or angiotensin II receptor antagonist.[2] [7]

Historical factors predisposing to atherosclerotic RAS include:[2]

- Multivessel coronary artery disease (CAD)
- Other peripheral vascular disease (PVD)
- Unexplained congestive heart failure (CHF)
- Refractory angina
- Dyslipidaemia
- Smoking (implicated in aetiology of both atherosclerotic and FMD types of RAS)[1] [11]
- Absence of family history of HTN - may be suggestive of RAS as a cause of HTN.[2] [12]

Examination

Given that RAS can only conclusively be diagnosed with imaging, suggestive findings on examination include:

- Hypertension on BP measurement
- Abdominal bruit: the finding of an abdominal bruit should raise the suspicion for the presence of RAS[2] [7]
- Other bruits: bruits in other vessels are frequent due to the common pathophysiology and high prevalence of co-existent PVD.[12]

General investigations

- Serum creatinine to estimate glomerular filtration rate.[1]
- Serum potassium: hypokalaemia or low-to-normal potassium may suggest activation of the renin-angiotensin-aldosterone system.[7]
- Urinalysis and sediment evaluation (to exclude glomerular disease): RAS, in the absence of co-existent diabetic nephropathy or hypertensive nephrosclerosis, is typically non-proteinuric without abnormalities in the urinary sediment.[4] [17]

Evaluation of secondary causes of HTN as indicated should be excluded and considered in the differential diagnosis: for example, aldosterone to renin ratio (ratio <20 excludes primary hyperaldosteronism).[7]

Choice of imaging

In addition to basic laboratory data, controversy remains as to what imaging modality is most appropriate. While ultrasonography offers a safe, non-invasive assessment, its sensitivity and specificity are low, and its use provides only indirect evidence of the presence of stenosis. Other non-invasive techniques (i.e., CT angiography or MR angiography) have a risk associated with the use of contrast media (radiocontrast nephropathy and nephrogenic systemic fibrosis, respectively). Conventional angiography, despite its procedural risk (e.g., atheroemboli, bleeding) and the risk of radiocontrast nephropathy, has the advantage of being able to determine the clinical significance of the lesions by measurement of the pressure gradient across a stenotic lesion, and the possibility of concurrently performing endovascular

therapy. Alternative imaging modalities that experts might consider in patients with chronic kidney disease (CKD) include non-contrast magnetic resonance angiography[18] [19] [20] and invasive angiography with carbon dioxide (CO2).[21] [22] [23]

It is recommended to start with a non-invasive imaging test in patients with a high clinical probability of RAS.

A patient's risk is determined by the clinician's index of suspicion, based on the patient's demographics (onset of HTN at age <30 years or >55 years), comorbid conditions (PVD, CAD, CVA), and clinical condition (HTN refractory to >3 antihypertensive agents).

If the results of non-invasive tests are inconclusive and the clinical suspicion for RAS is high, invasive testing is recommended.
[Fig-1]

[Fig-2]

[Fig-3]

[Fig-4]

Non-invasive imaging

It is reasonable to begin with a renal duplex ultrasound, followed if necessary by CT angiography, MR angiography, or a captopril renal scan. Non-contrast MR angiography sequences can be considered in patients with CKD.[18] [19] [20]

- Duplex ultrasound (sensitivity 84% to 98%, specificity 62% to 99%). Can identify discrepancy in kidney size, velocity of renal blood flow, and resistive index.[4] [14] [17] Ultrasound diagnostic criteria for significant renal artery stenosis are:[24]
    - Renal artery to aorta peak systolic velocity ratio (renal-aortic ratio) >3.5
    - Peak systolic velocity >200 cm/sec with evidence of post-stenotic turbulence
    - End-diastolic velocity >150 cm/sec (>80% renal artery stenosis) when present with a peak systolic velocity of >200 cm/sec
    - Renal resistive index >0.8 (sometimes used to predict response of blood pressure or kidney function for revascularisation).
- Gadolinium-enhanced magnetic resonance angiography (sensitivity 90% to 100%, specificity 76% to 94%).[2] [4] [14] [17] Noncontrast MR angiography sequences can be considered in patients with CKD.[18] [19] [20]
- CT angiography (sensitivity 59% to 96%, specificity 82% to 99%).[2] [4] [17] [25]
- Captopril renal scan (sensitivity 45% to 94%, specificity 81% to 100%) has a less relevant contemporary role because of its complexity, poor sensitivity, and the availability of easier and more accurate tests.[2] [4] [17] The American College of Cardiology Foundation/American Heart Association and the European Society of Cardiology/European Stroke Association/European Society of Vascular Surgery do not recommend captopril renal scan for diagnosing RAS.[14] [15]

Invasive testing

Conventional angiography:[2] [4] [17]

- The most sensitive and specific test for assessing anatomical narrowing of the renal artery.
- Also allows for therapeutic intervention at the same time.
- Requires arterial catheterisation and contrast utilisation.

Additional diagnostic modalities may be used during invasive angiography (such as the evaluation of pressure gradients, the use of pressure wires to evaluate lesion physiology, or intravascular ultrasound). Likewise, carbon dioxide angiography may be performed in specialised centres in patients with CKD.[21] [22] [23]

Risk factors

Strong

dyslipidaemia

- Cholesterol deposition in the vessel walls, followed by inflammation and progression of the cholesterol plaque.[1] [13]

smoking

- Favours endothelial inflammation and dysfunction.[1]
- Associated with both atherosclerotic and fibromuscular dysplasia (FMD).

diabetes

- Causes endothelial dysfunction; major cardiovascular risk factor.[1]

Weak

female sex

- Fibromuscular dysplasia (FMD) more frequent than in males. In addition, atherosclerotic RAS more likely to progress in this population.[1] [2]

History & examination factors

Key diagnostic factors

presence of key risk factors (common)

- Key risk factors include smoking, dyslipidaemia, and diabetes.

onset of hypertension (HTN) age >55 years (common)

- Suggestive of atherosclerotic RAS.[7] [14]

history of accelerated, malignant, or resistant HTN (common)

- Patients with RAS can present with severe, progressive, and/or difficult-to-control HTN, sometimes causing end-organ damage.[7] [14]

history of unexplained kidney dysfunction (common)

- Due to progressive stenosis or HTN-related end-organ damage.[7] [14]

history of multi-vessel coronary artery disease (common)

- Favours atherosclerotic RAS.[14]

history of other peripheral vascular disease (common)

- Favours atherosclerotic RAS.[2]

abdominal bruit (common)

- The finding of an abdominal bruit should raise the suspicion of the presence of RAS.[2] [7]

sudden or unexplained recurrent pulmonary oedema (common)

- Suggestive of RAS.[7] [14]

onset of HTN age <30 years (uncommon)

- Suggestive of fibromuscular dysplasia.[7] [14]

Other diagnostic factors

absence of family history of HTN (common)

- Suggestive of RAS.[2] [12]

other bruits (common)

- Bruits in other vessels are frequent due to the common pathophysiology and high prevalence of co-existent PVD.[12]

history of acute kidney injury after administration of ACE inhibitor or angiotensin II receptor antagonist (uncommon)

- This can be seen in some patients with bilateral RAS or RAS of a single functioning kidney, after starting an ACE inhibitor or angiotensin receptor blocker.
- Despite common belief that this class of medications is contra-indicated in this population, renin-angiotensin blockade is a proven therapeutic modality.[7] [14]

history of unexplained congestive heart failure (uncommon)

- Favours atherosclerotic RAS.[14]

refractory angina (uncommon)

- Favours atherosclerotic RAS.[14]

history of hypokalaemia (uncommon)

- Due to activation of the renin-angiotensin system.[7]

Diagnostic tests

1st test to order

Test	Result
serum creatinine • To estimate GFR.[7]	**normal or elevated**
serum potassium • Hypokalaemia or low-normal potassium may suggest RAS due to activation of the renin-angiotensin system.[7]	**low or normal**
urinalysis and sediment evaluation • Helpful in evaluating for glomerular source of kidney disease. In the absence of co-existent diabetic nephropathy or hypertensive glomerulosclerosis, RAS is not associated with proteinuria or abnormalities in the urinary sediment.[7]	**normal in the absence of diabetic nephropathy or hypertensive glomerulosclerosis**
aldosterone-to-renin ratio • Aldosterone-to-renin ratio <20 excludes primary aldosteronism as cause of hypertension and hypokalaemia or low-normal potassium.[7] • Testing requires discontinuation of anti-hypertensive medication.	**<20**

Other tests to consider

Test	Result
duplex ultrasound • Shows the renal arteries and measures flow velocity as a means of assessing the severity of stenosis.[1] [26] • Compares peak systolic velocity in the renal artery to that in the adjacent aorta. • Sensitive only for lesions with >50% reduction in vessel diameter, and unable to provide further quantification of stenosis. • Ultrasound criteria for significant renal artery stenosis: • renal artery to aorta peak systolic velocity ratio (renal-aortic ratio) >3.5 • peak systolic velocity >200 cm/sec with evidence of post-stenotic turbulence • the presence of an end-diastolic velocity >150 cm/sec is suggestive of >80% renal artery stenosis. [Fig-4]	**>50% reduction in vessel diameter**

Test	Result
gadolinium-enhanced MR angiography (MRA) • Visualises the renal arteries and peri-renal aorta.[1] • Use of gadolinium is recommended to be restricted in patients with stage 4 and 5 chronic kidney disease, due to nephrogenic fibrosing dermopathy. • MRA is not available to patients with pacemakers, some aneurysm clips, and other metal implants. • Blooming artifact can cause overestimation of stenosis if calcification is present. [Fig-2] [Fig-1]	**>50% reduction in vessel diameter**
CT angiography • Visualises the renal arteries and peri-renal aorta.[1] • Use of intravenous contrast challenging in patients with stage 3, 4, and 5 chronic kidney disease, due to risk of contrast-induced nephropathy. • Blooming artifact can cause overestimation of stenosis if calcification is present.	**>50% reduction in vessel diameter**
conventional angiography • Angiography is the most sensitive and specific test in the evaluation of RAS.[1] [2] • Possibility of performing intervention during the procedure. • Can measure pressure gradients across stenotic lesion to determine significance: • by measurement with a <5F catheter or with a pressure wire, a resting peak-to-peak gradient of ≥10 mmHg, or better a hyperaemic peak-to-peak gradient of ≥20mmHg, or a hyperaemic mean gradient of ≥7-10 mmHg is considered significant; hyperaemia can be induced with either dopamine or papaverine intra-arterially[27] [28] • a trans-stenotic-to-aortic pressure ratio of <0.9, as measured by a pressure wire across the stenotic lesion is considered significant, either at rest or with provocation with vasodilators.[29] • Angiography is an invasive procedure associated with known complications, including haematoma, pseudoaneurysm, renal artery dissection, atheroembolism, and acute kidney injury from intravenous contrast exposure. [Fig-3]	**>50% reduction in vessel diameter**
carbon dioxide (CO2) angiography • Alternative imaging modalities that experts might consider in patients with chronic kidney disease (CKD) include invasive angiography with carbon dioxide (CO2).[21] [22] [23] • CO2 angiography can be performed in specialised centres in patients with advanced CKD.	**>50% reduction in vessel diameter**
non-contrast magnetic resonance angiography • Alternative imaging modalities that experts might consider in patients with CKD include non-contrast magnetic resonance angiography.[18] [19] [20]	**>50% reduction in vessel diameter**

Test	Result
captopril radionuclide renal scan • Findings diagnostic of RAS include: a) delayed time to maximal radiotracer activity (TMax ≥11 minutes after captopril administration), b) significant asymmetry of peak activity of each kidney, c) marked cortical retention of the radionuclide after captopril administration, and d) marked reduction in calculated GFR of the ipsilateral kidney after ACE inhibition.[2] • To assess differential renal flow and kidney function between the two kidneys. • Useful in unilateral RAS, but limited in bilateral disease.[1] [8] • Has a less relevant contemporary role owing to its complexity, poor sensitivity, and the availability of other easier and more accurate tests.[2] [4] [17] The American College of Cardiology Foundation/ American Heart Association and the European Society of Cardiology/ European Stroke Association/European Society of Vascular Surgery do not recommend captopril renal scan for diagnosing RAS.[14] [15]	**delayed time to maximal radiotracer activity; significant asymmetry of peak activity of each kidney; marked cortical retention; marked reduction in calculated glomerular filtration rate (GFR)**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Essential hypertension	• No specific signs or symptoms. More frequently causes milder hypertension.	• Diagnosis of exclusion.
Acute kidney injury	• No specific signs or symptoms. Can be associated with difficult-to-control hypertension and abnormal volume status regulation.	• GFR is low. • Urinalysis and sediment evaluation may show proteinuria, haematuria, cells, casts, or crystals.
Renal artery dissection	• Difficult to differentiate clinically. • Although fibromuscular dysplasia places patients at a greater risk of renal artery dissection, spontaneous dissection of the renal artery or the aorta (involving the renal arteries) may cause severe HTN and loss of kidney function.	• Ultrasound, MR angiography, CT angiography, or conventional angiography will highlight an intimal flap.
Renal artery embolism	• History of other vascular disease, possibly history of catheterisation, although it may occur spontaneously.	• GFR may be reduced. • Eosinophilia may be present. • Lactate dehydrogenase level is commonly elevated. • Urinalysis and sediment evaluation may show WBCs and eosinophils.

Condition	Differentiating signs / symptoms	Differentiating tests
Chronic kidney disease	• Patients with chronic kidney disease typically have difficult-to-control HTN and volume status, which may mimic RAS. Furthermore, diabetes and hypertension are both causes of chronic kidney disease as well as RAS.	• GFR is typically reduced. • Urinalysis and sediment evaluation often show markers of kidney damage such as proteinuria or cells, casts, or crystals. • Kidney biopsy may demonstrate glomerular, tubular, or interstitial pathology.
Coarctation of the aorta	• Blood pressure different in arms and/or legs.	• Blood pressure in arms and legs demonstrates discrepancy. • Echo, MRI, and aortography may highlight coarct.
Primary hyperaldosteronism	• Resistant or accelerated HTN, adrenal adenoma.	• Plasma potassium may be low while urine potassium may be high. • Plasma aldosterone-to-renin ratio >20. • Adrenal CT may highlight a unilateral mass or bilateral gland enlargement. • Urine aldosterone is not suppressed after oral salt load. • Adrenal venous sampling demonstrates non-suppressible hormone levels.
Cushing's syndrome	• Moon face, buffalo hump, obesity, abdominal striae, possible history of corticosteroid administration.	• High morning plasma cortisol after 1 mg dexamethasone at bedtime. • Urinary cortisol levels are elevated. • Adrenal CT demonstrates gland enlargement. • Pituitary imaging may demonstrate adenoma.
Phaeochromocytoma	• Resistant or accelerated HTN, possibly episodic hypertension.	• Plasma-free metanephrines, urine metanephrines and catecholamines, and plasma normetanephrine are elevated. • Adrenal CT and scintigrams may demonstrate a mass.

Condition	Differentiating signs / symptoms	Differentiating tests
Vasculitis	• Usually with systemic symptoms (e.g., fever, weight loss), progressive kidney failure.	• Decreased GFR may be present. • Urinalysis and sediment may show proteinuria and haematuria. • Serological testing may be abnormal.

Diagnostic criteria

Severity[6]

- Moderate stenosis: 50% to 70% reduction in vessel diameter
- Severe stenosis: >70% reduction in vessel diameter
- Total occlusion: 100% reduction in vessel diameter.

Invasive measurements

- Catheter-measured hyperaemic peak-to-peak gradient of ≥20 mmHg or a mean gradient of ≥7-10mmHg[27] [28]
- Trans-stenotic ratio of <0.9 measured by pressure wire.[29]

Duplex ultrasound criteria[24]

- Ultrasound diagnostic criteria for significant renal artery stenosis are:
  - Renal artery to aorta peak systolic velocity ratio >3.5 (>50% stenosis)
  - Peak systolic velocity >200 cm/sec with evidence of post-stenotic turbulence (>50% stenosis)
  - Peak systolic velocity >200 cm/sec and end-diastolic velocity >150 cm/sec (>80% renal artery stenosis)
  - Renal resistive index >0.8 (sometimes used to predict response of blood pressure or kidney function for revascularisation).

Step-by-step treatment approach

The majority of patients with RAS have refractory or difficult-to-control hypertension (HTN), regardless of the aetiology (fibromuscular dysplasia [FMD] versus atherosclerotic). Many patients may already be taking multiple antihypertensive medications.

The addition of aspirin and high-intensity statin should follow as part of secondary prevention measures. Control of other risk factors (such as tobacco cessation or glycaemic control) is also recommended.

The role of percutaneous intervention remains controversial. It may be considered in patients with difficult-to-control HTN despite aggressive medical therapy, in the presence of test results confirmatory of RAS. Other indications include a rapidly declining level of kidney function or recurrent flash pulmonary oedema.

Surgical intervention is generally reserved for patients with concomitant vascular disease (e.g., abdominal aortic aneurysm [AAA]).

Only BP control with medicine, with or without intervention, will prevent or limit end-organ damage, such as progression of chronic kidney disease, and palliate some of the manifestations of this disease, such as refractory pulmonary oedema and angina. Patients with atherosclerotic RAS usually have concomitant cardiovascular disease, which should be aggressively treated.

Lifestyle modification recommendations for all patients

Low-salt diet:

- May help to improve BP in some patients.

Weight reduction:

- May help to improve BP in some patients.

Smoking cessation:

- This is an important part of management in both atherosclerotic and FMD RAS patients.[11] [14]

Optimisation of glycaemic control:

- Tight glucose control with goal HbA1c <53 mmol/mol (HbA1c <7%) is known to decrease the risk of microvascular complications.[30] However, tight glycaemic control has not been shown to decrease risk of macrovascular complications, and very tight control (HbA1c <42-48 mmol/mol [HbA1c <6%-6.5%]) may worsen it.[31] [32] Of antihyperglycaemic agents, only metformin has been associated with possible reduced macrovascular risk.[30]

Outline of antihypertensive medicines

Renin-angiotensin blockade with an ACE inhibitor or angiotensin II receptor antagonist is an attractive first-line antihypertensive strategy, as the actions target the mechanism of hypertension in RAS. Hypertension is a consequence of angiotensin II-related increase of systemic vascular resistance and stimulation of sodium retention, and ACE inhibitors have the potential to correct this state. However, some patients may not tolerate renin-angiotensin blockade, since GFR preservation may be dependent on high angiotensin II effect at the efferent arteriole to maintain intraglomerular pressure. It is recommended that BP, kidney function, and electrolytes be followed closely after initiating therapy.

First-line antihypertensives include captopril, enalapril, and angiotensin II receptor antagonists. The latter have not been studied in this population, but are generally considered equivalent to ACE inhibitors.[33] [34] [35] [36] Second-line preferences include thiazide or loop diuretics,[35] beta-blockers,[36] [37] calcium-channel blockers,[35] [38] or prazosin.[35] Third-line choices include clonidine,[35] hydralazine, methyldopa,[37] or minoxidil. Methyldopa and minoxidil are generally reserved for patients unresponsive to all other therapies. Patients may require referral to a HTN consultant (i.e., cardiologist or nephrologist).

Medical therapy versus intervention for atherosclerotic RAS

There is evidence that percutaneous vascular intervention plus medical therapy is no better than aggressive medical therapy alone. Evidence from the largest randomised RAS clinical trial to date (CORAL trial, n=947) suggests that percutaneous vascular intervention with stenting did not confer a significant benefit with respect to the prevention of clinical events when added to a comprehensive aggressive and multifactorial medical therapy regimen in people with atherosclerotic RAS. Clinical events in this trial were a composite end point of death from cardiovascular or renal causes, myocardial infarction, stroke, hospitalisation for congestive heart failure, progressive renal insufficiency, or the need for renal-replacement therapy. There were no specific subgroups with potential benefit.[39]

Therefore, renal artery percutaneous vascular intervention should be reserved for selected cases after careful assessment by a vascular and endovascular specialist.

Atherosclerotic RAS first-line treatment: antihypertensive + lifestyle modification + statin + aspirin

Antihypertensives

Statin:

- As atherosclerotic RAS is a type of vascular disease, statins should be considered for all patients. Although direct evidence is lacking for this recommendation, the high prevalence of concomitant peripheral vascular disease (PVD) and coronary artery disease (CAD) makes this recommendation reasonable. However, there are no published studies evaluating particular statin drugs, doses, or LDL cholesterol targets in this population. Weak evidence suggests that statins may slow the progression of atherosclerotic RAS.[40] [41]
- Any statin may be used, with a target LDL cholesterol <1.813 to 2.59 mmol/L (70 to 100 mg/dL).
- Baseline and periodic evaluation of LFTs are recommended.

Antiplatelet therapy:

- Aspirin should be considered for all patients with atherosclerotic RAS, because of the high likelihood of systemic cardiovascular disease.[14]

Atherosclerotic RAS second-line treatment: renal artery stenting

There are no data supporting renal artery stenting in asymptomatic patients in whom RAS is incidentally found.[2]

Current randomised data do not support routine percutaneous vascular intervention (angioplasty with or without stent deployment) in patients with atherosclerotic RAS. Percutaneous intervention was no better than medical therapy in decreasing clinical events such as death from cardiovascular or renal causes, myocardial infarction, stroke, hospitalisation for congestive heart failure, progressive renal insufficiency, or

the need for renal-replacement therapy in the CORAL trial.[39] Therefore, renal artery revascularisation should be considered only in selected individuals after careful evaluation by a vascular/endovascular specialist.

Guidelines suggest renal artery stenting could be considered in the following circumstances in selected individual patients, as discussed above:[3]
[Fig-3]

- Recurrent flash pulmonary oedema
- Stenosis of renal artery supplying single functioning kidney
- Refractory HTN on a multi-drug regimen (>3 medicines)
- Patients with RAS, uncontrolled HTN, and unstable angina
- Acute kidney injury on ACE inhibitors/angiotensin II receptor antagonist in patients with CHF.

Patients should be referred to an endovascular interventions consultant if percutaneous vascular intervention is considered. Current medical therapy should be continued and maximised. Transient addition of clopidogrel should be considered for dual antiplatelet therapy after the procedure.

Atherosclerotic RAS third-line treatment: surgery

Surgical reconstruction of the renal arteries in the setting of RAS is restricted to those patients undergoing major aortic reconstruction for another reason, such as AAA repair or correction of severe aorto-iliac occlusive disease.[14] Medical therapy should be continued with regular monitoring. Surgery is associated with 1% to 6% mortality.

Fibromuscular dysplasia first-line treatment: antihypertensives + lifestyle modification + percutaneous renal artery balloon angioplasty

Antihypertensives

Percutaneous renal artery balloon angioplasty:

- In addition to lifestyle modification and antihypertensive therapy, percutaneous renal artery balloon angioplasty should be considered first-line therapy, as it is often curative. Angioplasty has an initial technical success rate and 10-year patency rate of approximately 90%.[42]

Stenting:

- Is not indicated for initial treatment except in cases of procedural complications during percutaneous renal artery balloon angioplasty (i.e., renal artery dissection). It could be considered in the setting of re-stenosis.[14]
- Patients undergoing renal artery stenting require dual antiplatelet therapy (aspirin and clopidogrel) after the procedure.

Fibromuscular dysplasia second-line treatment: surgery

Surgical reconstruction of the renal arteries in the setting of FMD is restricted to those patients undergoing major aortic reconstruction for another reason. Surgical intervention may be necessary in complex disease that extends into the segmental arteries, and in cases of macroaneurysms.[14]

Medical therapy should be continued with regular monitoring.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Ongoing (summary)

Patient group	Tx line	Treatment
atherosclerotic RAS	1st	antihypertensive therapy + lifestyle modification
	plus	statin
	plus	antiplatelet agent
	2nd	renal artery stenting + continuation of medical therapy
	plus	post-stent clopidogrel
	3rd	surgical reconstruction of the renal arteries
fibromuscular dysplasia	1st	antihypertensive therapy + lifestyle modification
	plus	percutaneous renal artery balloon angioplasty
	2nd	surgical reconstruction of the renal arteries
▪ re-stenosis or percutaneous procedural complications	plus	renal artery stenting and dual antiplatelet therapy

Treatment options

Ongoing

Patient group	Tx line	Treatment
atherosclerotic RAS	**1st**	**antihypertensive therapy + lifestyle modification**

» The majority of patients with RAS have refractory or difficult-to-control hypertension (HTN). Thus, first-line treatment in this condition involves BP control to a target of <130/80 mmHg, given the high likelihood of concomitant cardiovascular disease.

» Renin-angiotensin blockade with an ACE inhibitor or angiotensin II receptor antagonist is an attractive first-line antihypertensive strategy, as their actions target the mechanism of HTN in RAS. However, it is recommended that BP, kidney function, and electrolytes be followed closely after initiating therapy.

» Patients may require referral to a HTN consultant (e.g., cardiologist or nephrologist).

» Some commonly prescribed antihypertensives are shown below. Doses should be started low and increased according to response.

» Lifestyle changes include weight loss, low-salt diet, and smoking cessation.

Primary options

» captopril: 12.5 to 25 mg orally three times daily

OR

Primary options

» enalapril: 10-20 mg orally once daily

OR

Primary options

» valsartan: 80-160 mg orally once daily

OR

Primary options

» losartan: 25-100 mg orally once daily

OR

Ongoing

Patient group	Tx line	Treatment

Secondary options

» hydrochlorothiazide: 25 mg orally once daily

OR

Secondary options

» furosemide: 40 mg orally once daily

OR

Secondary options

» atenolol: 50-100 mg orally once daily

OR

Secondary options

» amlodipine: 10 mg orally once daily

OR

Secondary options

» nifedipine: 30 mg orally (extended-release) once daily

OR

Secondary options

» prazosin: 2.5 mg orally once daily

OR

Tertiary options

» clonidine: 0.1 mg orally twice daily

OR

Tertiary options

» hydralazine: 10-50 mg orally four times daily

OR

Tertiary options

» methyldopa: 250-500 mg orally twice daily

OR

Tertiary options

» minoxidil: 5-40 mg orally once daily

Ongoing

Patient group	Tx line	Treatment
	plus	**statin**

» As atherosclerotic RAS is a type of vascular disease, statins should be considered for all patients.

» Although direct evidence is lacking for this recommendation, the high prevalence of concomitant peripheral vascular disease (PVD) and coronary artery disease (CAD) makes this recommendation reasonable. However, there are no published studies evaluating particular statin drugs, doses, or LDL cholesterol targets in this population.

» Weak evidence suggests that statins may slow the progression of atherosclerotic RAS.[40] [41]

» Any statin may be used, with a target LDL cholesterol <1.813 to 2.59 mmol/L (70 to 100 mg/dL).

» Baseline and periodic evaluation of liver function tests is recommended.

Primary options

» atorvastatin: 10-80 mg orally once daily

OR

Primary options

» fluvastatin: 20-80 mg/day orally (immediate-release) given in 1-2 divided doses

OR

Primary options

» lovastatin: 10-80 mg/day orally (immediate-release) given in 1-2 divided doses

OR

Primary options

» rosuvastatin: 5-40 mg orally once daily

OR

Primary options

» simvastatin: 5-40 mg orally once daily; increased risk of myopathy with 80 mg/day dose

	plus	**antiplatelet agent**

Ongoing

Patient group	Tx line	Treatment
		» Aspirin should be considered for all patients with atherosclerotic RAS.[14]
		Primary options
		» aspirin: 75-300 mg orally once daily
	2nd	**renal artery stenting + continuation of medical therapy**
		» There are no data supporting routine renal artery stenting in asymptomatic patients in whom RAS is incidentally found.[2]
		» Renal percutaneous vascular intervention appears to be no better than maximal optimal medical therapy.[39] However, in selected cases, previous guidelines suggest that renal artery stenting be considered in the following circumstances: refractory HTN on a multi-drug regimen (>3 medications); progressive chronic kidney disease; acute kidney injury on ACE inhibitors/angiotensin-II receptor antagonists in patients with congestive heart failure (CHF); recurrent flash pulmonary oedema; bilateral RAS; stenosis of renal artery supplying single functioning kidney; salvage therapy in recent-onset end-stage renal failure; patients with RAS, uncontrolled HTN, and unstable angina.[3] [Fig-3]
		» Patients should be referred to a peripheral vascular interventions specialist if revascularisation is considered. Current medical therapy should be continued and maximised. Transient addition of clopidogrel should be considered for dual antiplatelet therapy after the procedure.
	plus	**post-stent clopidogrel**
		» Patients undergoing renal artery stenting require dual antiplatelet therapy for a period of time as determined by the vascular specialist.
		» Patients should continue aspirin, with the addition of clopidogrel after the procedure.
		Primary options
		» clopidogrel: 75 mg orally once daily
	3rd	**surgical reconstruction of the renal arteries**
		» Surgical reconstruction of the renal arteries in the setting of RAS is restricted to those patients undergoing major aortic reconstruction

Ongoing

Patient group	Tx line	Treatment
		for another reason, such as abdominal aortic aneurysm (AAA) repair or correction of severe aorto-iliac occlusive disease.[14]
fibromuscular dysplasia	**1st**	**antihypertensive therapy + lifestyle modification** » The majority of patients with fibromuscular dysplasia have refractory or difficult-to-control HTN. Thus, first-line treatment in this condition involves BP control to a target of <140/90 mmHg. More aggressive BP control to <130/80 mmHg is indicated in the presence of concomitant cardiovascular disease. » Although no data support the use of a single agent or combination of drugs over another in fibromuscular dysplasia, the presence of compelling indication in comorbid conditions make antihypertensive selection important. » Furthermore, combination therapy with multiple agents is often necessary. » Renin-angiotensin blockade with an ACE inhibitor or angiotensin II receptor antagonist is an attractive first-line antihypertensive strategy, as their action targets the mechanism of HTN in RAS. However, it is recommended that BP, kidney function, and electrolytes be followed closely after initiating therapy. » Patients may require referral to a HTN consultant (e.g., cardiologist or nephrologist). » Some commonly prescribed antihypertensives are shown below. Doses should be started low and increased according to response. » Lifestyle changes include weight loss, low-salt diet, and smoking cessation. **Primary options** » captopril: 12.5 to 25 mg orally three times daily **OR** **Primary options** » enalapril: 10-20 mg orally once daily **OR**

TREATMENT

Ongoing

Patient group	Tx line	Treatment
		Primary options » valsartan: 80-160 mg orally once daily **OR** **Primary options** » losartan: 25-100 mg orally once daily **OR** **Secondary options** » hydrochlorothiazide: 25 mg orally once daily **OR** **Secondary options** » furosemide: 40 mg orally once daily **OR** **Secondary options** » atenolol: 50-100 mg orally once daily **OR** **Secondary options** » amlodipine: 10 mg orally once daily **OR** **Secondary options** » nifedipine: 30 mg orally (extended-release) once daily **OR** **Secondary options** » prazosin: 2.5 mg orally once daily **OR** **Tertiary options** » clonidine: 0.1 mg orally twice daily **OR** **Tertiary options** » hydralazine: 10-50 mg orally four times daily

Ongoing

Patient group	Tx line	Treatment
		OR **Tertiary options** » methyldopa: 250-500 mg orally twice daily **OR** **Tertiary options** » minoxidil: 5-40 mg orally once daily
	plus	**percutaneous renal artery balloon angioplasty** » Angioplasty, together with antihypertensive therapy, should be considered first-line therapy, as it is often curative. Angioplasty has an initial technical success rate and 10-year patency rate of approximately 90%.[42]
	2nd	**surgical reconstruction of the renal arteries** » Surgical reconstruction of the renal arteries in the setting of fibromuscular dysplasia is restricted to those patients undergoing major aortic reconstruction for another reason. Surgical intervention may be necessary in complex disease that extends into the segmental arteries, and in cases of macroaneurysms.[14]
■ **re-stenosis or percutaneous procedural complications**	plus	**renal artery stenting and dual antiplatelet therapy** » Stenting is not indicated for initial treatment of fibromuscular dysplasia except in cases of procedural complications during percutaneous renal artery balloon angioplasty (i.e., renal artery dissection). It could be considered in the setting of re-stenosis.[14] » Patients undergoing renal artery stenting require dual antiplatelet therapy (with aspirin and clopidogrel) following the procedure. **Primary options** » renal artery stenting **-and-** » aspirin: 75-300 mg orally once daily **-and-** » clopidogrel: 75 mg orally once daily

TREATMENT

Recommendations

Monitoring

- Close follow-up is required until adequate BP is achieved.
- Home BP monitoring is very helpful in assessing response to therapy.
- Periodic measurements (every 3 to 6 months) of creatinine and electrolytes are recommended. These are also important 2 to 4 weeks after adjusting doses of diuretics, ACE inhibitors, or angiotensin II receptor antagonists.
- If there are clinical changes or if the hypertension becomes uncontrolled, it is reasonable to obtain evaluation studies (invasive or non-invasive) to assess for progression of RAS.
- Surveillance Duplex ultrasonography may be useful in detecting re-stenosis early after the procedure (up to 1 year). Primary patency rates after stenting are 80% to 85%; secondary patency rates are 92% to 98%. Almost all occurrences of re-stenosis occur during the first year after stent implantation.[2]

Patient instructions

- Patients with RAS should understand that this is a chronic condition, likely to require close follow-up and multiple medications and/or interventions. They should be encouraged to be adherent with medications to achieve BP and lipid targets. [Vascular Cures: renovascular hypertension]
- Patients can keep a log of their BP at home. They can obtain an electronic cuff, which should be compared to the office standard sphygmomanometer once a year. They can check their BP about 3 times a day, at different times, recording these readings for review with their physician.
- In addition to being helpful in tailoring therapy and enlisting patients in their care, patients can be instructed to call their physicians if their BP increases significantly.

Complications

Complications	Timeframe	Likelihood
renin-angiotensin-associated orthostatic hypotension	**short term**	**low**
Occurs in approximately 10% of patients. May be associated with renin-angiotensin blockade.[48]		
renin-angiotensin-associated symptomatic tachycardia	**short term**	**low**
Occurs in approximately 10% of patients.[49]		
percutaneous intervention-associated inguinal haematoma	**short term**	**low**
Occurs in approximately 5% of patients undergoing percutaneous intervention.[54] [55]		
percutaneous intervention-associated retroperitoneal bleed	**short term**	**low**
Occurs in approximately 1% to 2% of patients undergoing percutaneous intervention.[56]		

FOLLOW UP

Complications	Timeframe	Likelihood
percutaneous intervention-associated femoral pseudoaneurysm or AV fistula	**short term**	**low**
Occurs in approximately 5% to 7% of patients undergoing percutaneous intervention.[57]		
percutaneous intervention-associated MI or stroke	**short term**	**low**
Occurs in approximately 0% to 5% of patients undergoing percutaneous intervention.[37] [53]		
progression of stenosis	**long term**	**medium**
Most patients with high-degree stenosis (>75% reduction in vessel diameter) tend to progress to total occlusion.[46] Approximately 10% of patients have unilateral stenosis >50%.[47]		
progression of chronic kidney disease	**long term**	**medium**
Overall there is no difference in kidney outcomes between patients treated medically versus those undergoing angioplasty.[43]		
percutaneous intervention-associated renal artery occlusion	**long term**	**low**
Occurs in approximately 2% of patients undergoing percutaneous intervention.[57] [58]		
percutaneous intervention-associated re-stenosis	**variable**	**medium**
Re-stenosis rates after percutaneous revascularisation, with or without stenting, range from 10% to 21%.[43]		
radiocontrast nephropathy	**variable**	**low**
Complication after CT angiogram or conventional angiography ± intervention. Patients with chronic kidney disease or diabetes, or who receive larger contrast doses, are at increased risk. Occurs in as many as 25% of these patients.[50] [51] Causes acute renal failure. May require dialysis in a small percentage of patients. N-acetylcysteine, bicarbonate, or saline IV prehydration and use of lowest possible doses of low-osmolar/iso-osmolar contrast agents should be considered in order to decrease the risk for radiocontrast nephropathy.[50] [51]		
percutaneous intervention-associated atheroembolism	**variable**	**low**
Reported in 1% to 2% of patients undergoing percutaneous intervention.[43] [52] [53]		

Prognosis

General outlook

Higher mortality and dialysis rates occur if there is worsening baseline kidney function, higher-grade stenosis, extensive coronary artery disease or peripheral vascular disease, or older age.[43]

Hypertension control

In bilateral RAS, angioplasty with stent deployment may be more effective than medical treatment alone regarding the goal of better BP control. Patients may require fewer medications for control of hypertension after stenting. An exceedingly small percentage of patients may be cured of hypertension.[43]

Kidney function

Current evidence indicates that kidney outcomes are no different with medical therapy or interventional therapy. However, kidney function improvement has only been reported in patients undergoing angioplasty.[43]

Medical therapy vs. interventional procedures: complications

Evidence does not support meaningful conclusions about relative adverse events or complications of percutaneous vascular intervention compared with medical treatment.[44] However, interventional therapy is associated with complications related to the procedure.

Patients undergoing renal artery percutaneous vascular intervention (angioplasty with or without stent deployment) require dual antiplatelet therapy (aspirin and clopidogrel) after the procedure. This carries a risk of bleeding.

Cardiovascular outcomes

There is no difference in cardiovascular outcomes between medical therapy and interventional procedures.[39] [44] [45]

Diagnostic guidelines

Europe

2017 ESC guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS)

Published by: European Society of Cardiology; European Stroke Organisation; European Society for Vascular Surgery **Last published:** 2017

Summary: Evidence-based guidelines that cover atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries, including the diagnosis of RAS.

North America

ACR appropriateness criteria: renovascular hypertension

Published by: American College of Radiology **Last published:** 2017

Summary: The American College of Radiology recommendations are focused on the different imaging modalities available for the diagnosis of RAS.

Clinical practice guidelines for the management of hypertension in the community: a statement by the American Society of Hypertension and the International Society of Hypertension

Published by: American Society of Hypertension; International Society of Hypertension **Last published:** 2014

Summary: RAS may be one of the aetiologies of secondary hypertension, along with chronic kidney disease, excessive aldosterone secretion, pheochromocytoma, and sleep apnoea. These guidelines provide a simple screening approach; to consider secondary causes of hypertension if specific measures fail to control blood pressure.

Expert consensus statement for renal artery stenting appropriate use

Published by: Society for Cardiovascular Angiography and Interventions **Last published:** 2014

Summary: Provides consensus-derived recommendations for diagnosing renal artery stenosis.

Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations)

Published by: American College of Cardiology; American Heart Association **Last published:** 2013

Summary: Provides a compilation of diagnostic recommendations from the following two guidelines: 2005 ACC/AHA guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic); and 2011 ACCF/AHA focused update of the guideline for the management of patients with peripheral artery disease (updating the 2005 guideline).

Treatment guidelines

Europe

2017 ESC guidelines on the diagnosis and treatment of peripheral arterial diseases, in collaboration with the European Society for Vascular Surgery (ESVS)

Published by: European Society of Cardiology; European Stroke Organisation; European Society for Vascular Surgery **Last published:** 2017

Summary: Evidence-based guidelines that cover atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries, including the treatment of RAS.

North America

2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8)

Published by: Eighth Joint National Committee **Last published:** 2014

Summary: This report takes a rigorous, evidence-based approach to recommend treatment thresholds, goals, and medications in the management of hypertension in adults.

Clinical practice guidelines for the management of hypertension in the community: a statement by the American Society of Hypertension and the International Society of Hypertension

Published by: American Society of Hypertension; International Society of Hypertension **Last published:** 2014

Summary: These guidelines provide recommendations for the management of hypertension in the community.

Expert consensus statement for renal artery stenting appropriate use

Published by: Society for Cardiovascular Angiography and Interventions **Last published:** 2014

Summary: Provides consensus-derived recommendations for the appropriate use of renal artery stenting.

Management of patients with peripheral artery disease (compilation of 2005 and 2011 ACCF/AHA guideline recommendations)

Published by: American College of Cardiology; American Heart Association **Last published:** 2013

Summary: Provides a compilation of treatment recommendations from the following two guidelines: 2005 ACC/AHA guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic); and 2011 ACCF/AHA focused update of the guideline for the management of patients with peripheral artery disease (updating the 2005 guideline).

Oceania

Chronic kidney disease (CKD) management in general practice (3rd edition)

Published by: Kidney Health Australia; The Royal Australian College of General Practitioners; Australian and New Zealand Society of Nephrologists

Last published: 2015

Online resources

1. Vascular Cures: renovascular hypertension *(external link)*

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Images

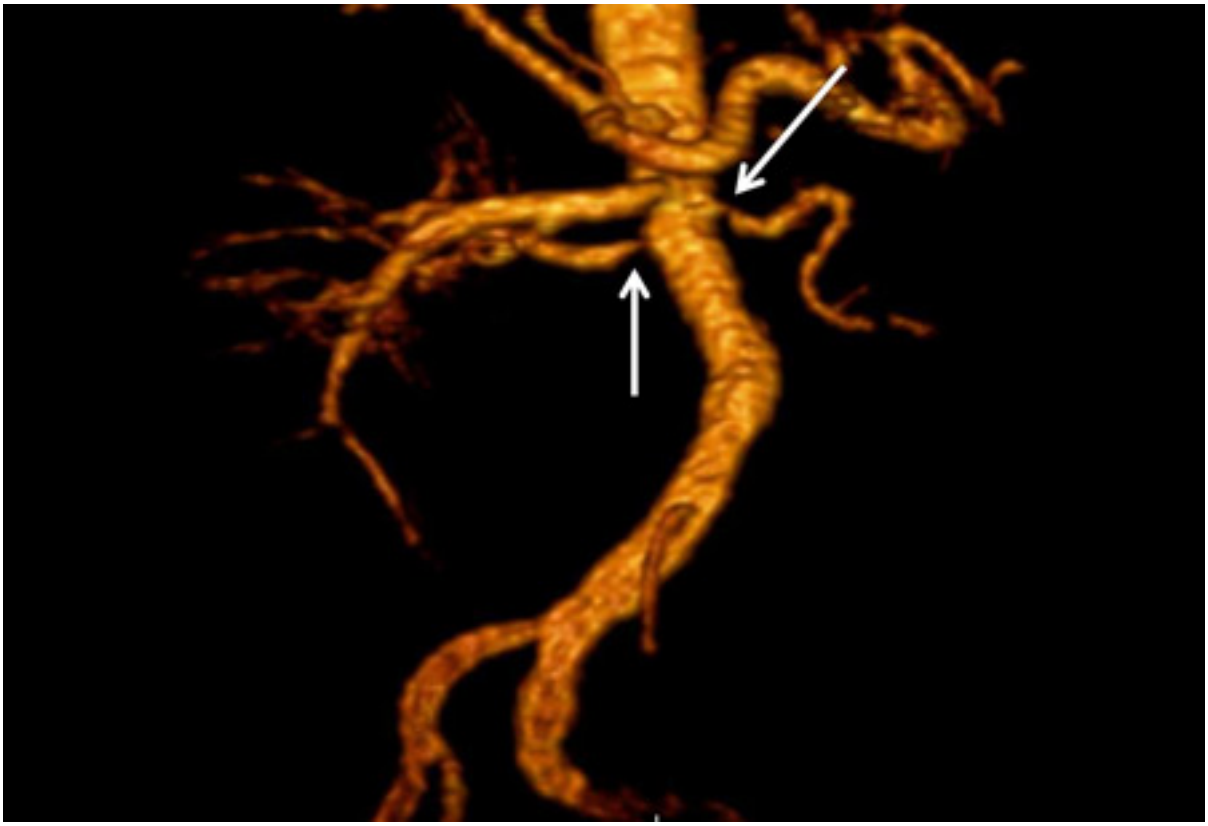

Figure 1: Magnetic resonance angiography (3-dimensional volume rendered reconstruction) in a patient with significant bilateral atherosclerotic renal artery stenosis. Arrows indicate proximal bilateral stenoses

Courtesy of David J. Sheehan, DO; Radiology Department, University of Massachusetts Medical Center and Medical School

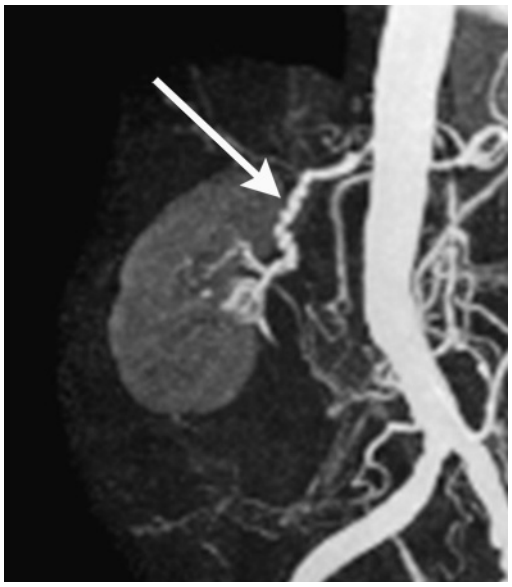

Figure 2: Magnetic resonance angiography (maximum-intensity projection) in a patient with fibromuscular dysplasia of the renal arteries. Arrow indicates the characteristic irregular contour in the right renal artery

Courtesy of Raul Galvez, MD, MPH and Hale Ersoy, MD; Department of Radiology, Brigham and Women's Hospital, Harvard Medical School

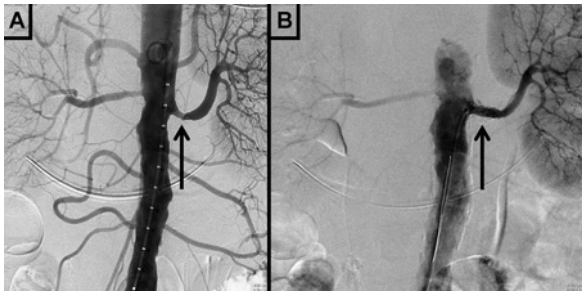

Figure 3: Digital subtraction angiography in a patient with significant atherosclerotic left renal artery stenosis. Panel A, prior to stent placement. Panel B, after successful stent deployment. Arrows indicate the site of stenosis and stent placement in their respective panels

Courtesy of Alvaro Alonso, MD and Scott J. Gilbert, MD

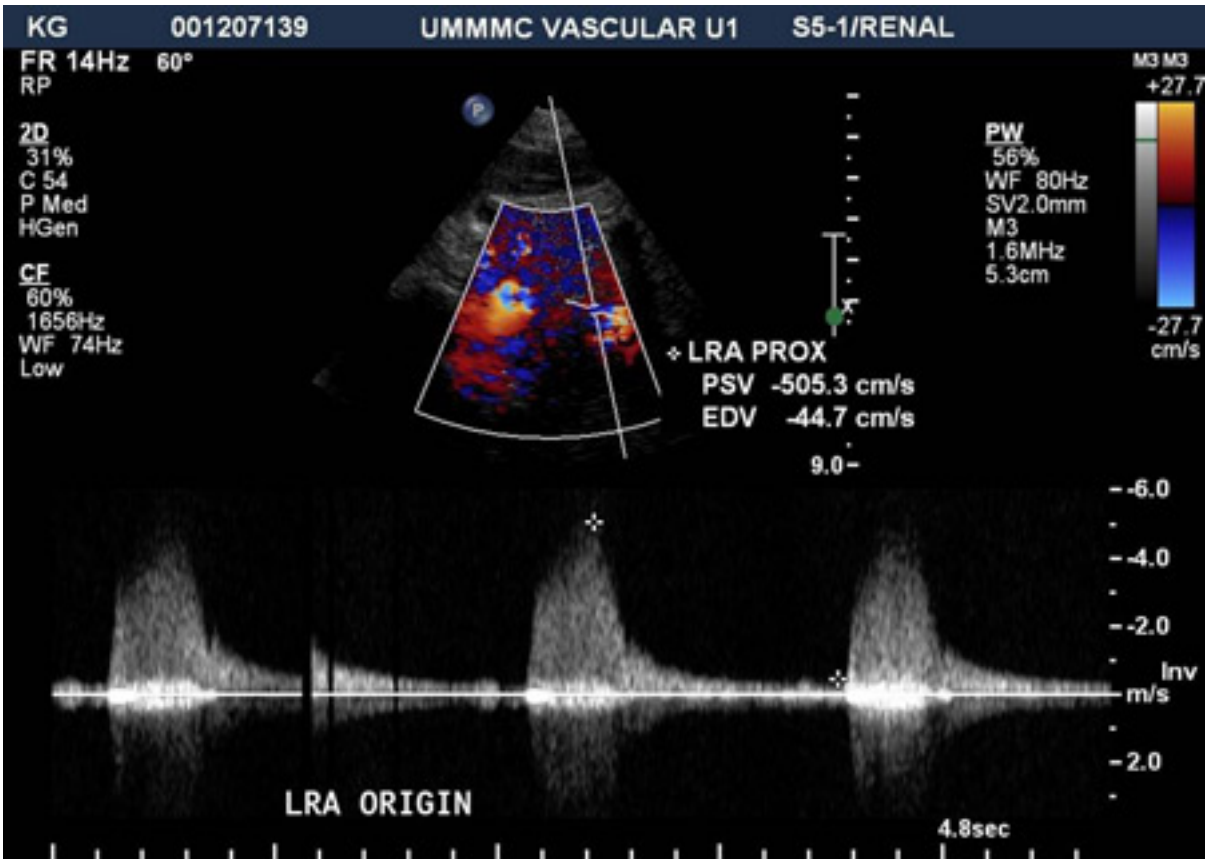

Figure 4: Colour and spectral Doppler of the left renal artery show colour disturbance due to turbulence, and an increased velocity of 50 m/second across a stenotic segment in the proximal left renal artery, consistent with significant renal artery stenosis

Courtesy of Denise Kush, RDMS, RVT; Vascular Laboratory, UMass Memorial Health Care

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