Chapter 26
Renovascular Hypertension

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Evaluating Patient

When should renovascular hypertension (RVH) be suspected?	In patients with: Refractory hypertension under age 30 New onset of severe/refractory hypertension after age 50 Abrupt worsening of controlled hypertension Hypertension with progressive renal failure Creatinine (Cr) rise over 20% with ACE inhibitors (AKI when put on ACE-I) Secondary hyperaldosteronism and resulting hypokalemia Flash pulmonary edema in patients with preserved LVEF Unilateral small kidney with difference >1.5 cm

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What is the definition of refractory hypertension?	Poorly controlled hypertension even on optimal medical management with three antihypertensive medications
What is the main underlying cause of RVH?	Renal artery stenosis (RAS) due to atherosclerosis (90%) or fibromuscular dysplasia (FMD) (10%)
How do patients with RAS usually present?	Asymptomatic, with RAS incidentally detected during unrelated angiographic imaging
What physical exam finding may be found in RAS?	Flank or abdominal bruit
What is a possible severe acute presentation of RAS?	Flash pulmonary edema
What is the best initial/ screening imaging study for evaluation of RAS?	Doppler ultrasonography (AHA/ ACC class I, LOE: B)
What are direct signs of RAS on Doppler?	Peak systolic velocity (PSV) > 180 cm/s Post-stenotic turbulence/bruit Renal artery PSV to aortic velocity ratio > 3.5
What are indirect signs of RAS on Doppler?	Tardus et parvus waveform distal to the stenotic lesion Prolonged acceleration time (> 0.07 seconds) Loss of early systolic peak distal to the lesion Discrepancy in resistive index (RI) before/after the lesion > 0.05
What is another noninvasive imaging modality that can help evaluate RVH when US findings are equivocal?	Computed tomographic angiography (CTA) (I, B)

What is a disadvantage of CTA (especially in this population), and what is an alternative test?	*Disadvantage*: Contrast-induced nephropathy in patient population with a high prevalence of ischemic nephropathy from stenosis *Alternative*: Magnetic resonance angiography (MRA) (I, B)
What is the gold-standard test for diagnosing RVH?	Digital subtraction angiography (DSA) (I, B)
What are other described diagnostic methods, and their AHA/ACC recommendation class/level of evidence?	Captopril renal scintigraphy (III, C) Selective renal vein renin sampling (III, B) Plasma renin activity (III, B) Captopril stimulation test (III, B)

High Yield History

About what percentage of patients with HTN have underlying RAS?	5% (0.5–10%)
What is the underlying pathophysiology of RAS causing RVH?	Decreased renal perfusion ➔ renin release by juxtaglomerular cells ➔ activates angiotensin II, causing: Efferent arteriole constriction to increase renal perfusion Systemic hypertension Aldosterone elevation leading to sodium/water retention and diastolic dysfunction
What is the definition of hemodynamically significant RAS (ACC/AHA and ACR/SIR)?	10% or greater mean translesional pressure gradient (and/or SBP gradient > 20 mmHg or > 10 mmHg mean gradient difference)

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What lab abnormality is associated with a worse prognosis in RVH?	Creatinine > 3.0
What are the demographics of RAS caused by atherosclerosis?	Older men
What are the demographics of FMD?	Women aged 30–50 years old
What subtype of FMD is associated with HTN?	Medial subtype, with intimal and adventitial subtypes being much less likely to cause HTN (< 15% combined)
What is the characteristic appearance of FMD?	"Beading" of the artery due to alternating stenoses and aneurysms
What findings are associated with FMD?	True and dissecting aneurysms Arteriovenous fistulas
What is another differential for RAS?	Noninflammatory vasculitis
What is the most common noninflammatory vasculitis subtype?	Medial fibroplasia
How is this treated?	Balloon angioplasty. Only pursue stenting if angioplasty fails or there are complications.
What are uncommon etiologies for RVH?	Renal artery aneurysm, Takayasu arteritis, neurofibromatosis, Iatrogenic or traumatic injury causing dissection, vessel injury damaging the intima causing thrombosis, retroperitoneal tumor encasement/compression, Williams syndrome, segmental arterial mediolysis, and midaortic syndrome

Indications/Contraindications

What is the gold-standard initial treatment for RVH?	Medical management including (class I, A): ACE inhibitors Angiotensin receptor blockers Beta-blockers Calcium channel blockers
When medical management fails, what are invasive treatment options?	Surgical or endovascular revascularization
What arteries are used for bypass in surgical revascularization?	Splenic artery for the left kidney and hepatic and gastroduodenal artery (GDA) for the right
When is surgery indicated over endovascular treatment?	FMD with segmental artery involvement or with macroaneurysms (I, B) Atherosclerotic RAS with multiple small renal arteries or early primary branching of the main renal artery (I, B) Atherosclerotic RAS with pararenal aortic reconstructions (e.g., after prior AAA treatment) Refractory/recurrent RVH after previous endovascular treatment
How is RVH treated endovascularly?	Atherosclerotic RAS: Angioplasty and stenting May be treated with angioplasty only if non-ostial location FMD: Angioplasty only

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What are the indications for renal vascular imaging or angiography (RAS screening) based on patient presentation (per the SIR, with ACA/AHA classes and levels of evidence)?	Onset of HTN before age of 30, especially without family history (I, B) Recent onset of significant HTN after the age of 55 (I, B) Accelerated, resistant, or malignant HTN (I, C) Sudden (flash) or recurrent pulmonary edema, especially with azotemia (I, B) Renal failure of uncertain cause, especially with a normal urinary sediment and less than 1 gram of protein per daily urinary output Coexisting, diffuse atherosclerotic vascular disease, especially in heavy smokers Acute renal failure precipitated by antihypertensive therapy, particularly angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (I, B) Idiopathic unilateral atrophic kidney (size difference >1.5 cm) (I, B) Unexplained renal failure (IIa, B)

What are potential indications for revascularization in RVH caused by RAS (ACC/AHA classes and levels of evidence)?	Asymptomatic patient with hemodynamically significant RAS (IIb, C) Hemodynamically significant RAS with (IIa, B): Accelerated HTN: sudden worsening of previously controlled HTN Resistant HTN: HTN that cannot be controlled (< 140/90, or SBP < 160 in patients over 60) on a maximally dosed triple-drug regimen including a diuretic Malignant HTN: HTN with end-organ damage including left ventricular hypertrophy, congestive heart failure, visual or neurologic disturbance, or advanced retinopathy HTN with an unexplained unilateral small kidney HTN with intolerance to anti-HTN medications Progressive CKD with (IIa, B): Bilateral RAS RAS to a solitary functioning kidney Chronic renal insufficiency with unilateral RAS (IIb, C) Hemodynamically significant RAS with cardiac destabilization syndrome (I, B), including: Recurrent, unexplained congestive heart failure Sudden, unexplained pulmonary edema Hemodynamically significant RAS and unstable angina (IIa, B) Acute, symptomatic, idiopathic renal artery dissection with new flank pain, hematuria, or accelerated HTN without underlying connective tissue disorder or other pathology (per ACR/SIR. No AHA/ACC recommendation)

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What are absolute contraindications to renal revascularization?	Hemodynamically nonsignificant stenosis
What are relative contraindications to renal revascularization?	Long-segment total occlusion Severely diseased aorta, as there is increased risk of embolization of the atheroma
When is stent placement indicated over balloon angioplasty?	Stenotic ostial atherosclerosis (within 1 cm of aortic lumen) Restenosis after prior treatment Postoperative (renal bypass, transplanted renal arteries) stenosis Highly eccentric renal artery stenosis Acute failure or complication of PTA including: Vessel recoil with possible collapse Complex dissections not responding to prolonged reinflation esidual stenosis > 30% or residual pressure gradient > 10% MAP Rupture or perforation (use covered stent)
How can you treat in-stent restenosis?	Options include PTA and re-stent.
When is stent placement relatively contraindicated?	Branch vessel disease Stent placement that would traverse renal artery branches Lesion length > 2 cm Renal artery diameter < 4 mm (can use coronary-sized stent) Unfavorable renal anatomy, without enough vessel length distal to proposed stenting to allow for future surgical bypass if needed Diffuse intrarenal vascular disease Noncompliant lesion Kidney size < 7 cm

What are indications for renal vein renin sampling?	To determine which patients may benefit from revascularization To determine the physiologic significance of RAS
What are contraindications to renal vein renin sampling?	Patients who are not candidates for revascularization Patients with occlusions of the renal vein/ICV or IVC filters
What can hinder accurate interpretation of renal vein renin sampling results?	Patients on chronic ACE inhibitors or beta-blockers not able to be safely taken off medication

Relevant Anatomy

How many renal arteries do patients commonly have?	One per side
What are important variations to normal renal artery anatomy?	Accessory renal artery arising from the aorta (can be unilateral or bilateral) Early branching of the renal artery, within 1 cm of the aorta
What lesion location lends more to atherosclerosis over FMD?	Ostial location (proximal third)
Which underlying cause is more likely in bilateral RAS, atherosclerosis or FMD?	Atherosclerosis

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What is the characteristic location of FMD?	Usually involving the mid to distal portion (the proximal artery may be involved, but rarely in isolation). This is often unilateral, with a preponderance for the right side over the left.

Relevant Materials

What kind of sheaths should be used for renal arterial interventions, especially in cases of atherosclerotic RAS?	Longer arterial sheaths (20–30 cm) are best as they help decrease the potential for plaque disruption during exchanges and manipulations. A 40 cm Flexor Ansel Sheath (Cook Medical) is often used during intervention.
What kind of wire and catheter can be used to cross an atherosclerotic RAS lesion?	Soft atraumatic wire (e.g., Bentson) and recurved catheter (e.g., Sos Omni Selective (AngioDynamics) or Simmons)
What balloon diameter should you use for renal arterial angioplasty?	Approximately 1% larger than estimated normal vessel diameter (not size of post-stenotic dilation)
What kind of stent is best for RAS, especially for ostial lesions, and why?	Balloon-expandable metallic stents, due to precision of placement
How do you choose stent size?	Adequately covers the lesion in length and with diameter matching normal (pre- and post-stenotic) vessel diameter (usually 1–2 cm length, 4–8 mm diameter)
What type of guidewires should be avoided and why?	Hydrophilic wires may cause perforation or dissection and may not provide enough support for stenting, though can be used to atraumatically cross lesions and then exchange for a working wire.

What do you use to measure a pressure gradient?	Lowest profile pressure wire (such as 0.014 in.)
What kind of catheter is ideal for renal vein renin sampling?	5 Fr. Cobra 2 catheter with a side hole made at the distal tip 2–3 mm from end hole
What catheters can help access the renal vein in difficult sampling cases?	Sidewinder or other recurve catheters

General Step by Step (DSA and Endovascular Revascularization)

Per the SIR, what are ideal coagulation parameters pre-endovascular intervention?	INR < 1.5 Platelets: Transfusion if below 50,000/L
Are prophylactic pre-procedural antibiotics recommended for endovascular revascularization?	No
How should you manage patients with chronic kidney disease (CKD) or risk factors for AKI or CKD (e.g., DM, MM, dehydration) periprocedurally?	Hydrate overnight with 0.45% saline with sodium bicarbonate at a rate of 100–150 cc per 4–12 hours. At least 1 hour of hydration. Use 30–50% diluted iodinated contrast or non-iodinated contrast such as carbon dioxide.
How should you manage a patient's hypertension prior to a renal revascularization procedure?	Discontinue long-acting antihypertensives and manage with short-acting antihypertensives instead, as able.

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Where should vascular access ideally be obtained?	Femoral, preferably on the right
Where should you obtain access if the patient has distal aortic occlusion or unfavorable renal artery angle?	Left brachial access, or radial access
What is the next step after obtaining access?	Diagnostic angiography starting with a flush aortogram and then selective renal angiogram
What is the best projection/ view angle for aortic disease and ostia?	LAO, about 20° for the right renal arter LAO, about 5–10° for the left artery
If disease is bilateral, on which kidney should you start intervening?	Start with the larger kidney, as disease is usually less severe and if cannot successfully treat that one, likely will not be able to treat either (this can also allow for a two-stage therapy, and at least help the patient in the interim).
What are techniques to decrease risk of embolization especially from atherosclerotic aorta?	"No touch" technique: 0.035 j wire placed alongside the guidewire inside guide catheter that is in the suprarenal aorta to lift the catheter tip off of the aortic wall "Sos flick" technique: Soft atraumatic guidewire 1–2 mm out of a SOS Omni Selective catheter, advance up the aorta with wire sticking out toward the direction of renal artery want, will "flick" in.
How do you prevent spasm of the renal artery prior to guidewire insertion?	Intra-arterial (IA) nitroglycerin (100–200 micrograms)

How can you provoke a pressure gradient to assess for need for revascularization?	50 ug/kg dopamine IA (this has the best evidence) 100–200 ug nitroglycerin IA 30–40 mg papaverine IA 1 mg isosorbide dinitrate IA
What do you do in the event of occlusive dissection or perforation?	Place a covered or uncovered stent.
How do you prevent thrombosis once you have crossed the stenosis?	Heparin IV: administer a 5000 unit bolus, followed by infusion of 750–1000 U per hour.
What is the target activated clotting time (ACT) for stenting/intervention?	2.5× baseline (~ 300+ seconds)
What is the target ACT when removing the femoral sheath at the end of the procedure?	< 180 seconds
How do you position the stent if the lesion is ostial?	Place stent to extend 1–2 mm into the aortic lumen.
How much stent overlap should there be if you are stenting in series (multiple stents)?	2 mm
How long do you keep the balloon inflated for angioplasty?	For 1 minute (or until/if patient has severe pain)
What do you do after completing angioplasty/ stenting?	Angiogram, avoiding recrossing the stenosis
What do you do if the angiographic result is not good or a significant pressure gradient still exists?	Upsize balloon to 1 mm larger than previous; repeat until good result.

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What must you do if endovascular revascularization has failed, and/or patient is planned to undergo surgical revascularization?	Angiography of possible donor vessels, most importantly the celiac access
What is the imaging modality of choice for follow-up after stent placement?	Renal Doppler ultrasound
What is the definition of technically successful endovascular renal revascularization (ACR/SIR)?	< 30% residual stenosis and a pressure gradient less than the selected threshold for intervention (< 10% and/or mean SBP gradient < 20 mmHg or 10 mmHg mean gradient difference)
What is the overall technical success rate of endovascular revascularization with stent placement in atherosclerotic RAS?	95% or greater
What labs should you monitor for 24 hours after the procedure?	Serum creatinine and BUN
How long should you monitor BP for?	At least 24 and up to 48 hours
What should you do if BP drops below normal levels?	Infusion of normal saline IV
What should you do if BP increases during or after the procedure?	Administer an ACE inhibitor such as captopril. Use other short-acting medicines if severely elevated.
Do any medications need to be continued post-procedurally?	If a drug-eluting stent was used, then aspirin 81 mg or another antiplatelet medicine must be used for 6 months.

When do most recurrences happen?	Within the first 8 months
What is the failure rate of primary stent placement requiring re-intervention?	~11%
What is the failure rate of repeat intervention on in-stent stenosis?	~20%
What are the risk factors for restenosis after stenting?	Stents dilated to less than 6 mm Female sex Age greater than 65 years Smoking
What is the technical success rate of angioplasty in FMD?	95% or greater
What is the primary patency of angioplasty-treated FMD?	69% at 4 years
What is the clinical response to angioplasty in patients with hypertension due to renal artery FMD?	22–39% cured and 31–59% improved/partial response
What are the definitions of clinical evaluation after revascularization?	*Cured*: BP< or = 140/90 without meds *Improved/partial response*: diastolic BP decreased by 10–15 mmHg or greater on the same or less meds, OR decreased in diastolic BP by 10–15 mmHg without medications (normal with meds) *Stable*: diastolic BP within 15 mmHg on the same or less meds *Failed*: diastolic BP unchanged on the same or less meds

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What are the definitions of evaluating renal dysfunction after revascularization?	*Improved*: decreased serum creatinine by 20% or more over baseline *Stable*: creatinine within 20% of baseline *Failure*: elevation of creatinine of 20% or more over baseline

Step by Step (Renal Vein Renin Sampling)

How should you manage patient's hypertension prior to renal vein renin sampling?	Off all antihypertensives for 2 weeks prior; most importantly, off beta-blockers and ACE inhibitors.
How can you increase the accuracy of renal vein renin sampling?	Captopril 60–90 minutes before procedure (increases renin secretion on affected side) Sodium depletion
What is a primary difference between renal vein renin sampling and renal endovascular revascularization, procedurally?	Venous puncture as opposed to arterial puncture
Where does left renal vein renin sampling occur?	Beyond the orifice of the left gonadal vein
Where does right renal vein renin sampling occur?	Close to the IVC, no gonadal vein drainage to worry about
Where do you obtain control samples from?	The infrarenal IVC
Can you use contrast in renal vein renin sampling? Why/why not?	No, contrast affects the production of renin, potentially altering the results.
What is the protocol for obtaining samples?	Obtain as closely together as possible (within 20 minutes), and transport to lab on ice.

Complications

What is the overall mean complication rate of endovascular intervention?	~14%
What is the most common type of complication?	Groin hematoma and puncture site trauma including hemorrhage, rupture, inadvertent venous puncture, and arteriovenous fistula (3–5%)
What are some possible complications at the angioplasty site?	Local thrombus Nonocclusive dissection (caused by guidewire or angioplasty) Arterial rupture
What is a risk of having balloon up too long or taking too long to deflate the balloon?	Thrombus formation and possible vessel occlusion
How do you manage local thrombus without significant dissection or vessel perforation?	Trial of local intra-arterial thrombolysis: 5 mg t-PA over 30 minutes, followed by 0.5 mg per hour for up to 24 hours
How do you manage arterial rupture?	Gently inflate balloon across the tear to tamponade. Deploy covered stent, as needed.
How do you manage non-flow-limiting dissections?	No management needed, common occurrence
How do you manage flow-limiting dissections?	Prolonged reinflation of a 1 mm undersized balloon or deployment of a covered stent
Which patients are at higher risk of vessel rupture in renal angioplasty?	Those on chronic steroid therapy
What are other risk factors for general complications and recurrence?	Current smokers Untreated hyperlipidemia

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What are some possible extra renal complications?	Emboli to extremities Puncture site complications Myocardial infarction
What is the rate of major complication (requiring surgery or prolonged hospitalization)?	3–11% (vs. 20% for surgery)
What are major complications, and their incidence?	Worsening of renal function due to contrast and/or multiple small infarctions by microemboli (3–5%) Occlusion of renal artery (2–3%) Segmental infarction and perinephric hematoma (1–2%) Need for surgical intervention such as nephrectomy and salvage (2%) Death (1%)
What is the 30-day mortality, and what are the causes?	< 1%. Caused by renal artery perforation, cholesterol embolization, ARF, and arterial access above the inguinal ligament with subsequent bleed
What is the 30-day surgical mortality?	Up to 5%
Which patients have a higher rate of complication with revascularization: those with FMD or atherosclerotic stenosis?	Atherosclerosis
What has been found to be the most significant factor in determining risk of complication?	Operator experience

Landmark Research

What have studies evaluating surgical versus endovascular revascularization for RAS found?	One RCT showed no difference in outcomes including blood pressure, patency, and complications, but demonstrated a longer hospitalization postsurgical repair. A large meta-analysis showed better long-term patency and decreased blood pressure from surgical repair, but demonstrated higher surgical mortality, especially in poor surgical candidates.
What about comparing stenting versus angioplasty alone (in atherosclerotic patients)?	One small RCT, plus one meta-analysis, demonstrated that stenting had a significantly lower risk of restenosis, with no difference in blood pressure or renal outcomes, making stenting more favored when considering endovascular intervention in these patients.
What were the findings of the STAR, ASTRAL, and CORAL trials comparing medical therapy alone versus medical therapy plus endovascular revascularization?	Multicenter randomized controlled trials which found no significant difference in progression or renal disease (STAR and ASTRAL) or cardiovascular events, blood pressure changes, and all-cause mortality (CORAL), between medical therapy alone and medal therapy with endovascular revascularization

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What are some significant limitations of these (STAR, ASTRAL, and CORAL) studies?	Poor selection criteria STAR: Patients were selected by inaccurate, non-angiographic imaging. No pressure gradient measured (no measurement of hemodynamically significance of RAS). Used 50% stenosis as threshold, not 70% as is more standard. ASTRAL: Patients were excluded based on subjective opinion of their physician. 40% of patients had < 70% stenosis (likely not hemodynamically significant). No pressure gradients measured. Intervened on patients with contraindications. CORAL: Patients without HTN enrolled (RAS may not have been clinically significant). Average % stenosis in treated group was < 70%. In most cases, these studies did not include high-risk patients (pulmonary edema, etc.). Poor technical outcomes in some, including higher complication rates and lower technical success rates than reported elsewhere, possibly due to inexperience of operators.
What is an argument in favor of the findings of the CORAL trial, as opposed to the others?	Selection criteria and decisions to intervene or not reflected current practice patterns at the time

What is an important finding possibly supporting revascularization in all of the above, and other similar, studies?	Patient who underwent endovascular revascularization had a decrease in the number of antihypertensives needed to control their blood pressure, and the procedure is usually associated with a low complication rate in the hands of experienced operators.
What were the findings of studies that have included high-risk patients (including flash or recurrent pulmonary edema, multiple high-risk comorbidities, and progressive renal failure), and what are their limitations?	Endovascular revascularization was associated with reduced risk of death/survival advantage over medical therapy alone. However, these studies have mostly been small, non-randomized, single-center studies.
What is the current state of endovascular revascularization in cases of renovascular hypertension from atherosclerotic RAS based on these studies, as summarized in multiple meta-analyses, review articles, and ACR-SIR practice parameter?	No strong evidence for endovascular revascularization over medical therapy alone in the majority of cases of renovascular hypertension. In a minority of severe cases of RVH, most notably in patients with flash or recurrent pulmonary edema, endovascular revascularization may be indicated. Operator experience level seems to be an important predictor of outcomes. More rigorous studies are needed, especially in high-risk patients.

Common Questions

What is the clinical hallmark of renovascular hypertension or HTN caused by RAS?	Poorly controlled HTN on optimal medical therapy with three different classes of drugs
What are the main causes of RAS?	Atherosclerosis FMD
What subtype of FMD is most associated with RVH?	Medial
What is the best screening and follow-up imaging modality for RAS?	Renal duplex ultrasound
What is the gold standard for diagnosis, and why?	DSA, ability to measure translesional gradients
What is hemodynamically significant RAS?	> 10% or 10 mmHg mean pressure gradient and > 20 mmHg systolic pressure gradient
What is the optimal treatment for RVH due to RAS?	Medical therapy including an ACE and/or an ARB
If intervening, what is the major difference in treating atherosclerotic versus FMD lesions?	Atherosclerosis: Stenting (usually with balloon angioplasty or balloon-mounted stents) FMD: Balloon angioplasty only

What presentation of RVH due to atherosclerotic RAS is the only indication with a class I recommendation for endovascular revascularization?	RVH causing cardiac destabilization, including flash and/or recurrent pulmonary edema Key: Classification of recommendations and level of evidence Class I: Benefit ≫ risk. Procedure should be performed. Class IIa: Benefit > risk. It is reasonable to perform the procedure. Class IIb: Benefit ≥ risk. Procedure may be considered. Class III: No benefit, or there is harm. Procedure is not helpful or may be harmful. Level A: Data from multiple RCTs or meta-analyses. Level B: Data from one RCT or from non-randomized studies. Level C: Limited data, only case studies or expert opinion.

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