

Renovascular Hypertension

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Renovascular Hypertension

- An important and potentially correctable cause of secondary hypertension
- Frequency varies with population studied
 - Less than 1% of mild to moderate HTN
 - Much more common with acute, severe or refractory HTN

Suggestive Clinical Findings

- Onset of HTN prior to 30 yo, particularly if no FH or other risk factors (ie obesity)
- Onset of severe (\geq stage II or 160/90) HTN after age 55
- Resistant HTN – not controlled on 3 drugs at therapeutic doses
- Acute rise in BP over previously stable baseline
- Malignant HTN – severe HTN with end organ damage (ARF, HF, neurologic change, papilledema, retinal hemorrhages)
- Acute rise in creatinine (\geq 30%) with ACE-I
- Moderate to severe HTN in patient with unexplained atrophic kidney or asymmetric kidney size (\geq 1.5cm)
- Moderate to severe HTN in patients with diffuse atherosclerosis (?bruit)
- Moderate to severe HTN with recurrent acute pulmonary edema
- ?Hyponatremia (unclear etiology)

Screening for Renovascular Hypertension

- Medical therapy with RAAS blockade (ACE-I, ARB, direct renin inhibitors) and diuretic (if necessary) is indicated in all patients with renovascular HTN.
- Often quite effective
- Therefore, do not screen for renovascular HTN unless discovery of a stenosis would alter treatment (ie an intervention would be performed)

Screening Tests for Renovascular HTN

- Renal arteriography is gold standard
- Noninvasive alternatives include
 - MRA
 - CTA
 - Duplex Doppler ultrasonography
- Other tests now felt to be not useful due to inferior accuracy
 - Captopril renal scintigraphy
 - Selective renal vein renin measurements
 - Plasma renin activity

Diagnostic Imaging Tests for Renovascular HTN

- MRA
 - Increasingly used as first line screening test.
 - Sensitivity approaches 100% with specificity 71-96% in populations at high risk of atherosclerotic disease. Less useful for patients with fibromuscular dysplasia.
 - Gadolinium contraindicated for $GFR < 30$
- CTA
 - Sensitivity and specificity in excess of 95% for detection of main renal artery lesions in patients with history suggestive of renovascular HTN.
 - Contrast must be given. With low GFR, CT contrast preferable to gadolinium.
- Duplex Doppler Ultrasonography
 - Noninvasive
 - Highly operator dependent
 - Can take up to 2 hours to perform
 - In good hands, sensitivity 85% and specificity 92%
- Test characteristics do vary with population studied.
 - Noninvasive testing most helpful in moderate risk patients.
 - For high risk patients, MR or CT reasonable alternative to angiography.
 - For patients at low risk for renovascular HTN for whom diagnosis needs to be pursued, angiography preferred (ie FMD).

Treatment of Renovascular HTN

- Diagnosis of renovascular HTN requires
 - Documentation of >75% stenosis
 - One or more of clinical features suggesting that stenosis plays an important role in elevated BP (see slide 3).
 - Discovery of an incidental stenosis at cath should not prompt revascularization
- Patients with atherosclerotic renovascular HTN have CAD equivalent and should have aggressive risk factor reduction according to published guidelines
- Three options
 - Medical therapy – indicated in all patients (if tolerated)
 - Angioplasty, generally with stenting
 - Surgery
- Revascularization, usually with stenting, warranted for >75% lesions and one of following
 - Resistant HTN
 - Malignant HTN
 - Inability to tolerate medications
 - Recurrent flash pulmonary edema

Revascularization vs Medical Therapy

- Several randomized trials have examined this issue, most notably ASTRAL
- Other than slight reduction in number of medications used, none demonstrated a significant benefit from stenting
- Limitations
 - Many patients had stenoses that were not clinically significant (50-70%).
 - Patients excluded if doctors felt they "definitely" needed revascularization
 - Primary outcome was progression of chronic kidney disease, not BP control
- In a meta-analysis of 1208 patients undergoing stenting, 4 deaths were reported and 12 renal artery perforations or dissections
- Surgery is indicated with
 - Multiple small renal arteries
 - Early primary branching of main renal artery
 - Need for aortic reconstruction near renal arteries.
 - Death rate 3-7% in these high risk patients.
- Revascularization probably not helpful in patients with advanced kidney disease (ie creat \geq 3)

Bilateral Renal Artery Stenosis and RAS associated CKD

- Medical therapy with ACE-I +/- diuretic safe in majority of patients with HD significant bilateral renal artery stenosis.
- ACE-I mediated reduction in GFR will occur but often not clinically significant
 - Only 5-10% will have large increase in Scr
 - First step would be to stop diuretic
 - A minority of patients are unable to have simultaneous BP control and stable renal function, and in these patients revascularization should be considered
- Theoretic concern of chronic renal hypoperfusion and atrophy in these patients with medical therapy. Risk of this is unclear.
- Chronic medical therapy of RAS also runs risk of progression of stenosis and eventual related renal failure.
 - 21 patients with bilateral RAS and 3 year follow-up
 - 17 stable renal function
 - 4 had 50% increase in Scr
 - 2 of those 4 on dialysis
 - But, overall mortality rate was 43% with MI and HF being major culprits
 - These patients often succumb of atherosclerotic disease of other organs

Conclusions

- Renovascular HTN is rare in overall population but fairly common in specific subgroups
- Aggressive risk factor modification is warranted (CAD equivalent)
- All patients should be treated with RAAS blockade +/- diuretic. This is generally well tolerated even with bilateral disease.
- Screening should only occur for patients in whom revascularization would be performed. MRA first choice, angiography gold standard.
- Revascularization (generally stenting) is of arguable benefit but probably plays some role in patients with relatively preserved renal function and difficult to control hypertension, perhaps with hypertension related complications