Renal Artery Stenosis: Diagnosis and Clinical Management

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GOALS

- Understand Renal Artery Stenosis (RAS) as an important sequela of atherosclerosis
- List three reasons to clinically suspect RAS
- Be familiar with the most up-to-date imaging and diagnostic modalities in RAS
- Consider the debate regarding medical vs. interventional management of RAS
RAS Introduction: Part 1 of 3

RAS can stand alone or it can exacerbate other disease processes, particularly HTN and renal failure.

Causes of RAS:
- Fibromuscular Dysplasia: 10%
- Atherosclerosis: 90%

RAS causes only 1% of all cases of HTN

RAS Introduction: Part 2 of 3

RAS can lead to renal failure

Safian et al NEJM 344: 6
See previous slide
RAS Introduction: Part 3 of 3

RAS can complicate pre-existing HTN

Atherosclerotic Disease
of Renal Artery (represents 90% of RAS)

→ Renin-Angiotensin System Activated
 Increased Vascular Oxidative Stress

→ Renovascular Hypertension

Acute/Severe HTN

Poorly-Controlled HTN

Well-Controlled HTN
(NB: Most patients with HTN have ESSENTIAL HTN. And most of the time, it responds to medical management)

In this presentation, we’re concerned with patients for whom RAS is complicating the management of their HTN.
RAS: A Key Player in Refractory HTN

- Mild Hypertension: RAS causes 1% of cases
- Acute, Severe, Refractory HTN: RAS causes 10-40% of cases
- In a study of 300 patients with well-controlled HTN:
  - 53% had normal renal arteries
  - 28% had <50% stenosis of one renal artery
  - 19% had >50% stenosis of one renal artery
  - 7% had >70% stenosis of one renal artery
  - 3% had bilateral disease
Meet Our Patient

Mr. W.R.
54 year-old male
PMH: HTN
Meds: ASA, Plavix, Norvasc (10 mg qd), Diovan (160 mg qd), Toprol XL (50 mg qd)
Former smoker
FH: DM
BP: 160-170 systolic
BUN/Cr: 1.1-1.2 mg/dl
Physical Exam: an abdominal bruit is appreciated

Should we suspect RAS?
Clinical Findings of RAS

- **Hypertension**
  - refractory to therapy with 3 or more drugs
  - abrupt onset before age 50—likely fibromuscular dysplasia
  - abrupt onset after age 50—likely atherosclerotic RAS
  - malignant HTN

- **Renal Abnormalities**
  - unexplained rise in BUN (azotemia)
  - unilateral small kidney
  - unexplained hypokalemia

- **Other Findings**
  - unexplained CHF or acute pulmonary edema
  - **abdominal bruit**, flank bruit, or both
  - severe retinopathy
  - peripheral or coronary vascular disease
If you suspect it clinically, how should you evaluate RAS?

- In choosing assessment and imaging modalities, consider the questions you hope to answer with the study.
Physiologic/Perfusion Studies

Guiding questions:
- What is overall kidney function?
- Is the lesion hemodynamically significant?

Tests
- Activity of renin-angiotensin system (renin levels, etc.)
- Serum creatinine
- Urinalysis
- Nuclear Medicine: Captopril Renography and Technetium-labeled DPTA
  - What is the differential flow velocity across each artery? What is the GFR in each kidney?

Drawbacks:
- lack of specificity (e.g. many disease processes cause increased creatinine)
- low predictive accuracy (i.e. studies do not indicate if re-vascularization would change clinical picture)
Vascular Studies

Guiding Question: What is the anatomic status of the lesion?

◆ **Gold Standard:**
  Contrast-Enhanced Angiography

◆ **Non-Invasive Studies:**
  – Duplex Ultrasonography
  – MR Angiography
  – CT Angiography (not discussed here, but increasingly popular)
Contrast-Enhanced Angiography

- Gold Standard, but no longer a diagnostic study– used for intervention
- Concerns: complications from procedure and contrast
  - consider use of gadolinium and visipaque as contrast agents in cases of renal insufficiency
Duplex Sonography

- **Inexpensive but significant drawbacks**
  - Operator-dependent
  - Time-consuming
  - Sensitivity: 81%
  - Specificity: 87%

- **Yields important functional information**
  - Hemodynamic information (what is the flow to each kidney?)
  - Predictive value with respect to revascularization (is lesion impeding flow?)
MR Angiography

- Sensitivity = 97%
- Specificity = 86%
- Fast, Operator-independent, 3-D Imaging
- Some limitations
  - Pacemaker incompatible
  - Inferior spatial resolution to interventional angiography (may over-estimate stenosis)
Our Patient’s MRA

Right Kidney

Minor Stenosis in right main renal artery

Left Kidney

75% Stenosis in Left Main Renal Artery

PACS, BIDMC
Patient MRA: A Different View

Stenosis in left main renal artery just proximal to take-off of minor inferior artery (at branch point)
Management Dilemmas in RAS

Although our patient presents with significant stenosis and clinical symptoms,

- **Incidental findings increasingly common**
  - What if RAS is found on abdominal MRA for a different indication in an asymptomatic patient?

- **Difficult to correlate progression of stenosis with clinical manifestations**
  - What if lesion was 50% this year and 60% the next? Does 10% progression justify intervention?

- **No good data comparing optimal medical management with revascularization interventions**

- **What to do?**
When faced with management dilemmas, consider the risks of disease progression and the goals of intervention

Progression of Atherosclerosis is Significant:
- Certain populations are at greater risk for worsening stenosis
- Although they rarely occlude, stenotic arteries narrow over time
- **Ischemic nephropathy associated with significant mortality**
  - Dialysis patients have 50% mortality over 3 years

Clinical consequences of intervention may not be significant:
- Will intervention improve renal function?
- **Will intervention change the medical management of the patient?**
RAS: An Approach to Intervention

- After assessing via MRA/CTA and before intervention, consider:
  - Clinical characteristics
  - Baseline renal function
  - Degree of asymmetry of flow

1) >50% Unilateral Stenosis
   or 10 mmHg gradient across lesion,

IF: 2) Bilateral Stenosis,
    or 3) Refractory HTN, Rising Cr,
    Recurrent Pulmonary Edema

- No intervention with advanced renal disease or chronic HTN
Intervention Strategies
Part 1 of 2

◆ Surgical
  – specific indications
    ◆ Fibromuscular Dysplasia (FD)
    ◆ Patient requires extensive reconstruction of aortic structures
  – recent data
    ◆ 2004 data available with long-term follow-up (11 years)
    ◆ 93% of those with FD had stable or improved renal function
    ◆ 71% of those with atherosclerotic disease had stable or improved renal function
Intervention Strategies

Part 2 of 2

- **Percutaneous Interventions**
  - **Balloon Angioplasty**
    - technically successful, but procedural risks
      - e.g. distal embolization, artery dissection
    - consider recurrence rates and effects on HTN
      - 15-42 percent re-stenose (inferior to stenting)
      - Unlikely to have long-term correction of HTN
  - **Stenting**
    - technically successful, avoids some risks of angioplasty
      - Stenting often repairs damage secondary to angioplasty
    - consider impact on renal function
      - Creatinine decline: 1.7 to 1.3 over four-year follow-up (N=1058)
      - Most lesions: 5-13% re-stenose (superior to angioplasty)
    - consider nature of stenotic lesion
      - No stenting in branch-vessel disease because stenting one vessel risks occlusion of the other
Which intervention did our patient undergo?

Angioplasty

Pressure Gradient: 100 mmHg

Recall that intervention warranted when pressure across stenosis exceeds 10 mmHg!

Remember: branch vessel disease is contraindication for stenting
Our Patient’s Angiography

After Angioplasty

Post-Procedure:
• BP: 140 systolic
• Cr: 1.1-1.2 mg/dl

5 mmHg
Recall our GOALS

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References (1 of 3)

**RAS: Pathophysiology, Natural History, Prevalence**


**Diagnostic Issues in RAS**

References (2 of 3)

*Diagnostic Issues in RAS Continued*


*Management Issues in RAS*

Management Issues in RAS Continued

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