RENAL ARTERIAL DISEASE

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INTRODUCTION

• Renin discovered in 1899, and Goldblatt defined relevant physiology in the 1930s.
• First successful treatment of renovascular hypertension occurred in 1938! (nephrectomy).
• First endarterectomy in 1954
• First pessimism by 1960!!! (“less than half of treated patients actually improve.”)
• In 2000, there were 21,600 renal revascularization procedures performed (Medicare only).

PATHOPHYSIOLOGY

• Physiology:
 • Obstruction to blood flow reduces perfusion and lowers intrarenal pressure
  • Perfusion:
    • Ischemic nephropathy – local hypertension more important than ischemia, per se
  • Pressure:
    • Release of renin, conversion of angiotensinogen to angiotensin I (and hence II by ACE) causing renovascular hypertension

DISCLOSURE

• I have no relationships to disclose.
• Endovascular intervention for renal and mesenteric disease is off-label and should be considered experimental.
**PATHOPHYSIOLOGY**

- Unilateral disease (Goldblatt “1 clip, 2 kidney”):
  - RENIN-DRIVEN. Continued renin release from bad kidney, but good kidney can excrete excess volume (renin inhibitors).
- Bilateral disease (Goldblatt “2 clips, 2 kidney”):
  - VOLUME-DRIVEN: Neither kidney can excrete excess volume; body settles down to a volume-overloaded homeostatic state (diuretics).

**ETIOLOGY**

- Atherosclerosis (“overflow” of aortic disease) (80%)
- Fibromuscular dysplasia (14%)
- Aneurysms
- Dissection and trauma (1%)
- Pediatric hypoplastic syndromes (3%)

**PRESENTATION**

- Hypertension
  - Young onset, sudden onset or worsening
  - Hypertensive crisis with flash pulmonary edema
  - Abdominal/flank bruit
  - Atherosclerotic risk factors
- Acute renal insufficiency after starting ACE/ARB
  - Loss of efferent vasoconstriction (compensation to maintain filtration pressure)
  - Incidental findings (aneurysm, FMD)
  - Trauma and dissection obvious

**PRESENTATION - IMAGING**

- Duplex
  - With skill, direction-independent
  - Classic criterion for greater than 60% stenosis – ratio of PSV in renal artery to aorta of >3.5
  - PSV >200cm/sec is abnormal, especially with poststenotic turbulence

**PRESENTATION - IMAGING**

- Resistive index: In theory a way to measure the intrinsic resistance of the renal parenchymal vessels – is this kidney likely to respond to elimination of the stenosis?
  
  \[
  \text{PSV} - \text{EDV} \quad \text{Values less than 0.7 to 0.8 suggest higher chance of improvement after revascularization.}
  \]

- Unfortunately, much better in theory than fact.
**PRESENTATION - IMAGING**

- CT or MR angiography excellent, however, for this very reason – rotatable
- However, contrast issues are significant in this patient population
  - GFR better than Cr

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**PRESENTATION – FXn?**

- Kidney length
- Pressure gradients
- Split function studies – relative function only
- Renal vein renin studies
  - Not terribly useful
TREATMENT

WHOM TO TREAT???

- Very unclear, especially today (2011).
- Classically: severe lesion with clear renovascular hypertension, bilateral lesions with ischemic nephropathy, pediatric patients.
- Hypertension: Unilateral or bilateral
- Ischemia: Theoretically must be bilateral, but hypertension itself causes nephropathy, so theoretically unilateral lesions can do it... but in this case it's probably beyond being responsive to revascularization.

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PATHOPHYSIOLOGY
PRESENTATION
TREATMENT
RESULTS
CONCLUSIONS

STAR (2003) and ASTRAL (2009) found no differences between medical treatment and stenting, but significant problems exist:
- ASTRAL
  - Mild lesions only (<70%, minimally symptomatic, physicians “weren’t sure whether intervention would help)
  - Inexperienced interventionalists (some performed 2 or fewer renal interventions yearly), high (10%) complication rate

ASTRAL, at least, did not include the population of patients (severe disease) that we are dealing with here – results only applicable to patients with very mild lesions (whom few of us would treat anyway).

CORAL – more severe lesions, ongoing, expect results in 2014

TREATMENT

WHOM TO TREAT???

- No evidence in support of treating incidentally-found lesions!

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General algorithm:
- Define anatomy and etiology
  - FMD/pediatric – early intervention
  - Severe hypertension not medically controllable, clear ischemic nephropathy, and those with “flash pulmonary edema” – early intervention
- All others, including incidentally found lesions (90%):
  - Medical treatment of BP
  - Atherosclerotic risk management
  - Close observation
TREATMENT

- Atherosclerotic stenosis
  - Endovascular is a good option – high success with low morbidity (but higher recurrence rates than surgery)
  - Lesion is usually “spillover” from aorta – hence
    - Primary stenting by most
    - Stent must protrude slightly into aortic lumen
  - Surgery (bypass or endarterectomy) works well in experienced hands – higher short-term risk but better long-term success rates.
TREATMENT

- FMD
  - Balloon angioplasty works well
- Dissection
  - Treat the primary problem, usually perfusion will “remodel”
  - Direct reconstruction/bypass
- Trauma
  - In theory, you have 30 minutes
  - Salvage is possible longer than this, but not if hours have elapsed

RESULTS

- Surgery (Wake Forest)
  - 720 reconstructions (10 years)
  - Mortality 1 to 3% (concomitant aortic surgery)
  - Morbidity 15 to 20%
  - Hypertension response: 85%
  - Function improvement: 70% off dialysis
  - Recurrence 4% (10% contralateral, unoperated upon arteries)
RESULTS

• Endovascular (meta analysis)
  • Approximately 2,000 patients (37 studies)
  • Mortality 0 to 3% (most zero)
  • Morbidity 0 to 43% (most access related)
  • Technical success >95%
  • Hypertension response: 60 to 80%
  • Function improvement: 25% off dialysis
  • Recurrence 5 to 66%

CONCLUSIONS

• TREAT:
  • Severe hypertension (uncontrolled by medical management or associated with cardiopulmonary compromise) in patients with severe renal artery stenosis
  • True ischemic nephropathy (so identified in collaboration with nephrology/cardiology)
  • Pediatric patients, those with FMD

• DO NOT TREAT:
  • Incidentally-found lesions (EVAR!)
  • Mild stenoses
  • Stenoses in patients with easily controllable hypertension

CONCLUSIONS

• Recent studies discourage intervention, but they did not include the categories of patients usually considered for intervention by most thoughtful physicians – ASTRAL does not apply to patients with severe lesions and severe sequelae.

• Endovascular intervention is associated with less short-term risk but has higher recurrence rates.
  • Surgical bypass is more dangerous in the short term, but has the best long-term outcomes.
CONCLUSIONS
• If discovered incidentally:
  • Repair all in pregnant or premenopausal women
  • Probably repair all that are 2.0 cm or enlarging
  • Define anatomy, talk to your colleagues – method of repair is individualized and especially if ruptured, have a low threshold for nephrectomy, splenectomy, and/or simple ligation.