Renovascular Disease

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R4 UBC Urology
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Scope of discussion

- RVH & IN
  - Atherosclerosis
  - Fibrous lesions
- RAS in children
- Renal artery aneurysm
- AVM
Sources

- Campbell’s 9th Ed., Ch 36
- Schrier’s Atlas of Diseases of the Kidney, VIII, Ch3
- AUAUS – V.16(24), 22(39), 25(33)
- Literature Review

Case#1

- 79 yo woman POD#8 AAA repair, no prior urologic Hx
- Intraop:
  - clamping above renal arteries
  - Prolonged renal ischemic time & clot in renal arteries
- Postop:
  - Initial course uneventful
  - Spontaneous brisk BRB via foley 24:00 hrs POD#8
  - Hemodynamically unstable: 60/40, HR 85
  - Mild R flank discomfort
  - Code Blue called, definitive therapy organized
Case #2

- 45 yo healthy woman, no prior urologic Hx
- Spontaneous painless gross hematuria
- Initially coincided with menses
- GP on day #7 of bleeding → Abx for presumed UTI
- Developed clot ret’n → ER → in&out & home
- Multiple repeat episodes clot retention → urology consulted as o/p
- Cystoscopy = normal bladder, clot, no bleeding
- U/S – “normal”
- Hb drifting 120→93
- CTA established Dx & definitive therapy organized

Historical Context

- 1827 – Bright associates ‘fullness’ of pulse, dropsy, & proteinuria with ‘hardening of kidneys’
- 1856 – Traube – high BP suspected
- 1874 – Mohomed – HTN assoc. c renal dz
- 1898 – Tigerstedt & Bergmen describe pressor substance from kidneys ‘renin’
- 1934 – Goldblatt – cause diastolic HTN in dogs by clamping renal arteries
- 1937-8 – several cases resolved HTN c Nx described
- 1939 – Goormaghtigh & Grimson – JG cells hypergranular in those with HTN (?secreting renin)
- 1940 – Page, Helmer, Braun-Menendez – renin=enzyme, angiotensin described
- 1950 – Blake, Pitts, Duggan – dog renal a. constriction → decr. Na & water excretion
- 1964 – Howar & Connor use above with ureteral catheters to determine “ischemic kidney”
- 1950’s & 60’s – advent of angiography & increased appreciation of RAAS system
Hypertension defined

- BP is a continuum
- Generally accepted def’n = 140/90
  - Untreated HTN associated with increased mortality (Lew, 1973, Insurance viewpoint in HTN Manual)
- WHO def’n = 160/95
- Dx requires several repeated readings
- Children age 12-15: upper limit N = 130/80

Renovascular HTN Defined

- Renovascular disease in pt with HTN ≠ RVH
  - Lesion must be proven to be functionally significant → reduces RBF to the point of activating renin release
RAAS System

- Renin-Angiotensin-Aldosterone System
- 1ª role = maintain tissue perfusion (esp. in setting of hypotension)
- Triggers:
  - True hypotension/hypovolemia
  - Perceived hypovolemia
    - RAS
    - CHF
    - Liver dz
RAAS System

5 Mechanisms of Renin release:
- Macula Densa
  - thick asc. Limb of LoH
  - Decreased distal tubule salt delivery → renin release
- Baroreceptor Mechanism
  - JG cells of aff. arteriole sense stretch: ↓stretch → hyperpolarization → ↓intracellular Ca → renin release
- Neural Mechanism
  - B-adrenergic receptors in JG cells respond to sympathetic stimulation → renin release

RAAS System

5 Mechanisms of Renin release:
- Endocrine/Paracrine
  - Stimulators: PG-E₂, PG-I₂, Arachadonic acid
  - Inhibitors: AII, endothelin, vasopressin, ANP
- Intracellular Mechanisms
  - Stimulators = Agents increasing adenylate cyclase activity (cAMP ²nd messenger in renin release): B-agonists, PG-E₂, PG-I₂, dopamine, histamine, PTH
  - Inhibitors = agents increasing intracellular Ca: AII, vasopressin, adenosine
RAAS System

Renin-angiotensin-aldosterone system

Renovascular Hypertension
Pathophysiology of RVH

- **Goldblatt (1934):** 2K, 1C ; 1K, 1C models

  **STUDIES ON EXPERIMENTAL HYPERTENSION**

  I. THE PRODUCTION OF PERSISTENT ELEVATION OF SYSTOLIC BLOOD PRESSURE BY MEANS OF RENAL ISCHEMIA†

  BY HARRY GOLDBLATT, M.D., JAMES LYNCH, M.D., RAMON F. HANZAL, Ph.D., and WARD W. SUMMERVILLE, M.D.

  (From the Institute of Pathology, Western Reserve University, Cleveland)

  PLATES 23 AND 24

  (Received for publication, December 1, 1933)

  - 2K, 1C = unilateral RAS with N contralat kidney
  - 1K, 1C = RAS of solitary kidney, RAS of transplant kidney, RAS with parenchymal damage to contralat kidney
  - B/L RAS = mixed bag

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*b* Afferent impulses from the ischemic kidneys may, in some way, bring about increased output of some internal secretion which, by peripheral or central action, may effect general vasoconstriction, and thus raise the blood pressure.

*(c)* There may be an accumulation or new formation of some substance, or there may occur a disturbance of chemical equilibrium between substances present in the blood which may effect a pressor action like that of a hormone.
Pathophysiology of RVH

- 2 Kidney, 1 Clip Model
  - ↑ renin
  - RAAS activation
  - ↑ AII
  - Systemic VC, HTN
  - ↑ Aldosterone
  - Na & H2O ret’n
  - ↓ renin
  - “pressure natriuresis”
  - Na & H2O loss

- 1 Kidney, 1 Clip Model
  - ↑ renin
  - RAAS activation
  - AII → HTN
  - Na & H2O ret’n
  - Hypervolemia & HTN
  - ↓ renin, AII
Ischemic Nephropathy

- Deterioration of renal function secondary to RAS
- Cause = chronic hypoperfusion of total functional renal mass (bilateral RAS or RAS in solitary kidney)
- Ischemic injury occurs when renal compensatory mechanisms overcome
Ischemic Nephropathy

- ↓RBF → RAAS activation → AII → efferent vc
  → attempt to maintain GFR
- 70% stenosis → ↓GFR, ↑Creat

Campbell’s 9th Ed.

Ischemic Nephropathy

- Pathophysiology underlying injury poorly understood
- Patchy tubular necrosis (most prominent)
- ↓glomerular size
- Thickened Bowman’s capsule
- Glomerulosclerosis (local & generalized)
- JG apparatus hypercellularity
- Renal atrophy
Renovascular Lesions

Classification of RV Lesions

- Atherosclerotic dz (70%)
- Fibroplasias
  - Intimal
  - Medial
  - Perimedial
- Fibromuscular hyperplasia (FMH)
- Other – RAA, MAoS, Periarterial fibrosis, Post-traumatic
Atherosclerosis

Scope of epidemic:
- Leading cause of illness & death in western countries
- 2002 = 74,626 Canadian deaths d/t CVDz
  - 32% of all male deaths
  - 34% of all female deaths
- 84% CVDz deaths d/t CADz, stroke, PVDz
- CVDz cost = >$18,000,000,000/year
- MRI data – Aortic ASO in “healthy” Framingham heart study participants
  - 38% women; 41% men

Sources:
Stats Canada, Causes of Death 2002
Public Health Agency of Canada, Economic Burden of Illness in Canada, 1994
Jaffer et al, ATVB, 22:849, 2002
Atherosclerosis

Risk Factors
- Hypercholesterolemia
- Low HDL level
- Hypertension
- Diabetes
- High Lipoprotein-a level
- Smoking
- Hyperfibrinogenemia
- Hyperhomocysteinemia
- Physical inactivity
- Obesity
- Male gender
- FHx or premature CADz
- Post-menopausal state
- ACE polymorphism
Atherosclerosis

- Accounts for 70% of all renovascular lesions
- Usually proximal 2cm renal a.
- Lesion involves intima
- 2/3 lesions are eccentric plaques
- Dissecting hematomas common → thrombosis of entire vessel

Initial angiogram

5 years later...
Atherosclerosis

The natural history of atherosclerotic and fibrous renal artery disease

Martin J. Schreiber Jr.1, Andrew C. Novick2, and Marc A. Pohl1

1 Department of Hypertension and Nephrology, Desk A 101, and 2 Department of Urology, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44195-5942, USA

- Retrospective study; pts on medical Rx
- 85 patients with ARAS on angiogram
- Sequential angiograms 3-172 mos post-Dx
- 44% = progressive obstruction
- 16% progressed to complete obstruction
- Most progress within 2 years of Dx

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Atherosclerosis

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- Progression related to initial degree obstruction
  - Mild (<50%) - 31% (5% complete)
  - Moderate (50-75%) – 47% (10% complete)
  - Severe (>75%) – 39% to complete occlusion
- Worsening renal f’n related to ARAS progression (54% vs. 25% in stable dz)
- BP didn’t rise with ARAS progression (despite rising Creat & diminishing renal size)
Incidence of ESRD 2° ARAS unknown

- Estimates = 1.4-2.1% of pts starting dialysis (based on reported cause for ESRD) Fatica, 2001
- Studies of CTA/MRA in patients starting dialysis:
  - Van Ampting (2003): 27% with ARAS
  - Uzu (2002): 50% with ARAS

Incidence of ESRD 2° ARAS

- Scoble et al, 1989
- All patients presenting to nephrology dept with difficult HTN, asymmetric kidneys, unexplained renal failure, systemic VDz “investigated” for ARAS
- 6% all new HD pts (14% those >50yo) had ESRD 2° ARAS

Atherosclerosis & ESRD

- Patients do very poorly on dialysis
  - Novick (1994): 76% 1-year mortality rate
  - Mailloux (1988): 12% 5-year survival rate

Atheroembolism

- Cholesterol embolism
- Usually older pts with severe aortic ASO
- Contributes to progressive decline in renal f’n (obstruction $\rightarrow$ inflammation $\rightarrow$ fibrosis)
- Often spontaneous vs. precipitated
- Precipitating factors: surgical manipulation of Ao, angiography, thrombolytics
- Mgt = supportive, remove traumatic factor, stop anticoagulation, control hypertension, IHD prn
Fibrous Arterial Lesions

- Intimal Fibroplasia
- Medial Fibroplasia
- Perimedial Fibroplasia
- Fibromuscular Hyperplasia (FMH)

Intimal Fibroplasia

- 10% fibrous lesions
- Children & young adults
- Circumferential collagen in internal elastic lamina
- Cx = dissection in outer half of media & aneurysmal dil’n
- Smooth, focal stenosis
- Proximal or mid-artery or branches
- Non-operative mgt = progression = obstruction & ischemic atrophy of kidney
Medial Fibroplasia

- 75-80% fibrous lesions
- Women 25-50yo
- Often bilateral
- May be generalized d/o
- Int. Elastica → focally & variably thinned
- Alternating thinned media & thickened media
- Microaneurysms & saccules
- “string of beads”
- Distal 2/3 of main & branches
- Minimal collateral circulation

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1 Department of Hypertension and Nephrology, Desk A-101, and 2 Department of Urology, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44195-5002, USA

- 66 patients with MF observed with serial angiography
- Progressive RAS in 33% (no total occlusion)
- Renal f’n decline & decr. Renal size rare
- Conclusions:
  - risk of losing renal f’n low in patients with MF managed medically
  - If decline renal f’n/size occurs → consider alternate Dx
Perimedial Fibroplasia

- Women 15-30yo ("girlie dz")
- 10-15% fibrous lesions
- Only in renal a.
- Tight ring of collagen in outer media
- "bead" no larger than artery diameter
- Extensive collaterals
- Dissection/thrombosis rare
- Ischemic renal atrophy universal

Fibromuscular Hyperplasia

- Children & young adults
- Very rare: 2-3% fibrous lesions
- Hyperplasia of smooth muscle & fibrous tissue
- Concentric thickening of vessel wall
- Angiographically indistinguishable from IF
- Natural Hx = progressive vascular obstruction
Clinical Clues to RVH Dx

- Age of onset: <30yo ~FD; >55yo ~ASO
- +FHx suggestive of essential HTN
- Sudden onset, short duration
- Difficult to control HTN (despite 2-3 meds)
  - Accelerated, malignant HTN; HTN crisis
  - HTN associated with pulmonary edema
- Systemic ASO
- Gradual impairment of renal f’n
- Flank pain (dissection, segmental infarction)
- Smoking = risk factor for ASO

Clinical Clues to RVH Dx

- Physical Exam
  - Severe HTN
  - Abdominal/renal bruits (biphasic)
  - Severe hypertensive retinopathy (Gr. III or IV)
  - Generalized ASO
Clinical Clues to RVH Dx

- Laboratory findings
  - Mild to severe proteinuria (multiple other causes)
  - Azotemia in presence of generalized ASO
  - Azotemia after medical control of BP (suggests perfusion-dependent renal f’n)
  - Hypokalemia in absence of diuretic use

Indications for Screening

- Older patients with HTN and any or all of the following:
  - Evidence of generalized ASO
  - Decrease in size of one or both kidneys
  - Renal insufficiency (esp. in absence of identifiable cause)
  - Progressive azotemia after meds for BP control
  - CHF
RVH & IN Diagnosis

- IVP
- Peripheral Plasma Renin Activity (PRA)
- Captopril test
- Renal vein renins
- Captopril renography
- Duplex U/S
- MRA
- CTA
- Angiography = gold standard

Angiography

- Gold Standard
- Diagnostic & therapeutic
- Disadvantages:
  - Invasive
  - Contrast use
  - Expensive
  - Complications: bleeding, hematoma, dissection, thrombosis, distal embolisation of plaque/cholesterol
Digital Subtraction Angiography

- Subtracts bone & soft tissue from image
- Reduced contrast load & catheter size (vs. conventional angiography)
- Improved contrast resolution
- Reduced spacial resolution

IVP

- Delayed contrast in calyces of affected kidney (key feature)
- Size discrepancy b/w kidneys >1.5cm
- Delayed concentration of contrast in collecting system
- Retention of contrast in nonobstructed system
- Notching of pelvicalyceal system by collateral vessels
Peripheral Plasma Renin Activity

- Screening test for RVH; no value for IN
- No anatomic information
- No antihypertensives x2 wks (limiting)
- PRA level indexed to Na intake
- Blood collection at noon after 4 hours ambulation
- Limitations:
  - 80% sensitivity; 84% specificity (20% FN)
  - 16% pts with essential HTN will have incr. PRA

Captopril Test

- Measurement of PRA before & 1 hr after captopril dose (25mg)
- Patients with RVH have higher rise in PRA than those with EH
- Stop all ACEI & diuretics 1 week prior
- Sensitivity = 75% (poor screening test)
- Specificity = 89%; NPV = 95% (good for ruling out RVH)
Renal Vein Renins

- Net renin secretion = $R_{RV} - R_{IVC}$
- IVC renin = Ao renin = PRA
- Ischemic kidney = positive value
- Normal/less ischemic kidney = 0
- Confirmatory test = $R_{RV} 50\% >$ than PRA

Captopril Renography

- ACEI → loss of GFR → scintigraphic changes
- Well hydrated patient, liberal salt intake
- MAG3 = best imaging characteristics (vs. DTPA & OIH)
- Diagnostic Criteria (Nally, 1991)
  - Size asymmetry on scintigraphic images
  - >11 min time to maximum activity
  - Asymmetry of time to peak activity b/w sides
  - Marked cortical retention of tracer
  - Marked decreased GFR on affected side
- Sensitivity = 90-93%; Specificity = 93-98%

Campbell's 9th Ed, p.1170
**Duplex U/S**

- **Basis of Dx = altered flow pattern beyond stenosis**
  - Systole = turbulent jet; PSV >180 cm/sec; RAR >3.5
  - Diastole = diminished flow
- **Sensitivity: 75-98%; Specificity: 90-100%**
- **Disadvantages:**
  - Operator dependent
  - Fat, bowel gas
  - Complete occlusion based on small kidney c no flow
  - No functional significance

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**MRA**

- **Advantages**
  - Noninvasive, no radiation
  - No iodinated contrast
  - Low technical failure rate
  - Can obtain functional information (RBF & GFR)
- **Disadvantages**
  - Poor image quality vs. angio
  - Visualizes proximal arteries only
  - Contraindications: magnetic implants, claustrophobia
- **Sensitivity: 88-93%; Specificity: 90-98%**

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Strandness, Am J Kid Dis 1994, 24:674
Mollo et al, Eur J Vasc Endovasc Surg 1997, 14:305

Thornton et al, Eur Radiol 1999, 9:930
CTA

- 2mm slices, arterial phase, 3D recon
- Advantages: ~cheap, widely available
- Disadvantages
  - Large contrast bolus required
  - ~ Poor definition beyond mainstem renal a.
- Sensitivity: 90-96%; Specificity: 97-99%

Diagnostic Modalities Compared

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<tr>
<th>Modality</th>
<th>Sensitivity</th>
<th>Specificity</th>
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<tr>
<td>Peripheral Renin Activity</td>
<td>80%</td>
<td>84%</td>
</tr>
<tr>
<td>Captopril Test</td>
<td>75%</td>
<td>89%</td>
</tr>
<tr>
<td>Captopril Renography</td>
<td>90-93%</td>
<td>93-98%</td>
</tr>
<tr>
<td>Duplex US</td>
<td>75-98%</td>
<td>90-100%</td>
</tr>
<tr>
<td>MRA</td>
<td>88-93%</td>
<td>90-98%</td>
</tr>
<tr>
<td>CTA</td>
<td>90-96%</td>
<td>97-99%</td>
</tr>
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Diagnostic Modalities Compared

US, CT, and MR Evaluation of Accessory Renal Arteries and Proximal Renal Arterial Branches

Ethan J. Halpern, MD, Levon N. Nazarian, MD, Richard J. Wechsler, MD
Donald G. Mitchell, MD, Eric K. Ouwater, MD, David C. Levin, MD
Geoffrey A. Gardner, Jr, MD, Harold I. Felman, MD

- 56 patients → all conventional angio
- 45 DUS; 52 CTA; 28 MRA (no gad.)
- 28 accessory arteries, 21 proximal branches

Halpern et al, Acad Radiol 1999, 6:299

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<table>
<thead>
<tr>
<th>Modality</th>
<th>No. of Patients</th>
<th>No. Identified with Arteriography</th>
<th>No. Detected with Other Modalities</th>
<th>No. of Additional Arteries Suggested</th>
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<td>4</td>
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<tr>
<td>MRI</td>
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<td>15</td>
<td>11</td>
<td>9</td>
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Halpern et al, Acad Radiol 1999, 6:299
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Detection of Accessory Arteries and Proximal Branches with US, CT, and MR Imaging

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Halpern et al, Acad Radiol 1999, 6:299

Approach to Dx

Ischemic Nephropathy

Strong Suspicion RAS

Low-Mod Suspicion RAS

DSA

Non-Invasive Anatomic

Poor Renal Function

DUS or MRA

Good Renal Function

CTA

Campbell's 9th Ed.
Approach to Dx

Renovascular HTN

Strong Suspicion RAS

Low-Mod Suspicion RAS

DSA

Captopril Renography

Positive Test

Poor Quality Test

Negative Test

DUS/MRA/CTA

“To date, anatomic confirmation of the disease and treatment planning still require intra-arterial angiography”
(Campbell’s 9th Ed.)

Who should be treated?

- Renovascular HTN:
  - Fibrous Disease
    - Medial Fibroplasia → aggressive medical mgt 1st
    - Intimal & Perimedial Fibroplasia → early surgical mgt
      - Main Renal a. lesions only → angioplasty
      - Branch lesions → surgical revascularization
  - Atherosclerotic Dz → aggressive medical mgt.
Who should be treated?

- **Ischemic Nephropathy:**
  - Factors for consideration:
    - Severity & extent of RAS
    - Renal function
    - Renal histopathology

Who should be treated?

- **Ischemic Nephropathy: Anatomic factors**
  - Indication for Rx
    - >75% stenosis affecting entire renal mass (b/l dz or dz with solitary kidney)
    - Novick et al, 1987
      - 161 patients with ARAS b/l or in solitary kidney
      - 58% improved f’n, 31% stable f’n, 11% declining f’n

Novick et al, JAMA 1987, 257:498
Who should be treated?

- **Ischemic Nephropathy: Anatomic factors**
  - **Indication for Rx**
    - >75% stenosis affecting entire renal mass (b/l dz or dz with solitary kidney)
  - **No indication for Rx**
    - Unilateral RAS with normal contralateral kidney
    - Dean et al, 1991
      - 53 patients with IN (41 b/l RAS, 12 u/l RAS)
      - GFR improved in pts with b/l RAS, no change if u/l

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Who should be treated?

- **Ischemic Nephropathy: Anatomic factors**
  - **Indication for Rx**
    - >75% stenosis affecting entire renal mass (b/l dz or dz with solitary kidney)
  - **No indication for Rx**
    - Unilateral RAS with normal contralateral kidney
    - **Gray-zone**
      - Unilateral RAS with contralateral kidney with parenchymal dz
      - Complete arterial occlusion
Who should be treated?

- Ischemic Nephropathy: Anatomic factors

  - Complete arterial occlusion: favourable factors
    - Angiogram → collaterals filling distal arterial tree
    - Kidney size >9cm
    - Function on renogram or IVP
    - Elevated lateralizing renal vein renins
    - Well preserved glomeruli on renal Bx

  Scheffet et al, JU 1980, 124:184
  Zinman & Libertino, JU 1977, 118:517

Who should be treated?

- Ischemic Nephropathy: Renal function
  - Predictors of functional recovery/stabilization
    - Mild – moderate renal dysf’n at presentation
      - Mercier et al, 1990
      - 9 year retrospective data
      - 48 revascularizations in 43 patients
      - Mean age = 61.8 yrs; Mean f/u = 35.1 mos

  Mercier et al, Ann Vasc Surg 1990, 4:166
Who should be treated?

Ischemic Nephropathy: Renal function
– Predictors of functional recovery/stabilization
  - Mild – moderate renal dysf’n at presentation
  - Rapid decline in renal f’n with antihypertensives

Textor, Arch Int Med 1983, 143:2208-11
Who should be treated?

- **Ischemic Nephropathy: Renal function**
  - Predictors of functional recovery/stabilization
    - Mild – moderate renal dysf’n at presentation
    - Rapid decline in renal f’n with antihypertensives
    
     ![Graph](image1)

Who should be treated?

- **Ischemic Nephropathy: Renal function**
  - Predictors of functional recovery/stabilization
    - Mild – moderate renal dysf’n at presentation
    - Rapid decline in renal f’n with antihypertensives
    - Rapid decline in GFR
    - ?ESRD d/t bilateral renal a. obstruction
      - Kaylor, 1989
      - 12 patients on HD (1-13 mos)
      - All had improved renal f’n and no more HD req’d

Kaylor et al, JU 1989, 141: 486
Who should be treated?

- **Ischemic Nephropathy: Histopathology**
  - ?salvageable function vs. irreversible damage
  - Useful with Creat >350
  - Unfavourable findings:
    - Arteriolar nephrosclerosis (most common)
    - Atheroembolic disease
    - Widespread glomerular hyalinization

  Campbell’s 9th Ed.
  Zinman & Libertino, JU 1977, 118:517

Treatment Options

- Nephrectomy or partial Nx (RVH)
- Surgical Revascularization
- Percutaneous Transluminal Angioplasty
- Endovascular Stenting
Pre-op W/U

- Beware the vasculopath
  - Cardiovascular w/u – incl. EKG, stress test, +/- cardio Cx & coronary angiogram
  - Cardiac revasculariz’n takes precidence > renal
  - Consider carotid doppler +/- endarterectomy

- Those with fibrous dysplasia usually young & otherwise healthy

Nephrectomy & Partial Nx

- Limited role
  - Irreversible renal parenchymal dz
    - Severe arteriolar nephrosclerosis
    - Severe renal atrophy
  - Uncorrectable renovascular lesions
Surgical Revascularization

- Aortorenal bypass
  - Autogenous graft (saphenous v., hypogastric a.)
  - PTFE
- Endarterectomy
- Extracorporeal microvascular repair & autotransplantation
- Splenorenal (L) or Hepatorenal (R) bypass

Minimal reported morbidity/mortality for fibrous dysplasias

Atherosclerotic dz
  - Operative mortality: 2.1-6.1%

Technical success rate (thrombosis & restenosis) = >90%
Surgical Revascularization

- RVH Outcomes
  - Cure = <140/90, no meds
  - Improve = DBP drop of 10-15 OR normotensive with meds
  - Failure = everyone else

- Fibrous Dysplasia:
  - Cure: 50-60%
  - Improve: 30-40%
  - Failed ~10%

- Atherosclerosis: ~30% cure, ~60% improve

| Table 7–5. RESULTS OF SURGICAL REVASCUULARIZATION FOR ATHEROSCLEROTIC RENOVASCULAR HYPERTENSION |
|-----------------|-----------|-----------|-----------|
| Series          | Patients (N) | No. Cured | No. Improved | No. Failed |
| Van Bockel et al, 1987 | 105 | 19 (18%) | 64 (61%) | 22 (21%) |
| Novick et al, 1987 | 188 | 55 (30%) | 110 (60%) | 18 (10%) |
| Liberato et al, 1988 | 86 | 38 (44%) | 44 (51%) | 4 (5%) |
| Hansen et al, 1992 | 332 | 22 (13%) | 116 (35%) | 14 (10%) |

Campbell’s 9th Ed.
Surgical Revascularization

- Ischemic Nephropathy Outcomes:
  - ~50% improve
  - ~33% stable
  - Remainder deteriorate

<table>
<thead>
<tr>
<th>Series</th>
<th>Patients (N)</th>
<th>No. Improved</th>
<th>No. Stable</th>
<th>No. Deteriorated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Novick et al. 1987</td>
<td>161</td>
<td>91 (56%)</td>
<td>50 (31%)</td>
<td>18 (11%)</td>
</tr>
<tr>
<td>Hallett et al. 1987</td>
<td>91</td>
<td>20 (22%)</td>
<td>48 (53%)</td>
<td>23 (25%)</td>
</tr>
<tr>
<td>Hansen et al. 1992</td>
<td>70</td>
<td>34 (49%)</td>
<td>23 (36%)</td>
<td>11 (15%)</td>
</tr>
<tr>
<td>Branderberg et al. 1992</td>
<td>40</td>
<td>22 (55%)</td>
<td>10 (25%)</td>
<td>8 (20%)</td>
</tr>
<tr>
<td>Libertino et al. 1992</td>
<td>91</td>
<td>43 (49%)</td>
<td>31 (35%)</td>
<td>15 (16%)</td>
</tr>
</tbody>
</table>

Table 7-6. RESULTS OF SURGICAL REvascularization FOR atherosclerotic ischemic nephropathy

Campbell's 9th Ed.

Angioplasty

- Mechanism
  - Fractures atherosclerotic plaque
  - Stretches arterial wall → tearing media & adventitia

- Complications (5-10%)
  - Transient decline renal f’n (contrast)
  - Complications of femoral a puncture
  - Intimal dissection/flap (Rx = stent)
  - Thrombosis (Rx = thrombolytic)
Angioplasty

- RVH 2° Fibrous Dysplasia: Outcomes
  - Usually performed without stent placement
  - Primary modality of Rx for FD
  - Most cases of restenosis successfully retreated

<table>
<thead>
<tr>
<th>Study</th>
<th>Technical Success</th>
<th>Follow-up</th>
<th>Cured</th>
<th>Improved</th>
<th>Failed</th>
<th>Restenosis</th>
<th>Major Complications</th>
<th>Minor Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sis et al, 1983</td>
<td>87%</td>
<td>16 mo</td>
<td>52%</td>
<td>29%</td>
<td>19%</td>
<td>6%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Martin et al, 1985</td>
<td>82%</td>
<td>16 mo</td>
<td>56%</td>
<td>25%</td>
<td>17%</td>
<td>11%*</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Lentinger et al, 1989</td>
<td>88%</td>
<td>20 mo</td>
<td>41%</td>
<td>47%</td>
<td>12%</td>
<td>0%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Gilge et al, 1989</td>
<td>90%</td>
<td>15 mo</td>
<td>50%</td>
<td>15%</td>
<td>12%</td>
<td>12%*</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Seiter et al, 1991</td>
<td>100%</td>
<td>30 mo</td>
<td>39%</td>
<td>10%</td>
<td>2%</td>
<td>10%</td>
<td>2%</td>
<td>14%</td>
</tr>
<tr>
<td>Hain et al, 1993</td>
<td>9 mo</td>
<td>36 mo</td>
<td>50%</td>
<td>30%</td>
<td>15%</td>
<td>20%</td>
<td>0</td>
<td>10%</td>
</tr>
<tr>
<td>Giannino et al, 1994</td>
<td>35%</td>
<td>6 mo</td>
<td>50%</td>
<td>40%</td>
<td>15%</td>
<td>15%</td>
<td>0</td>
<td>10%</td>
</tr>
<tr>
<td>Jensen et al, 1995</td>
<td>57%</td>
<td>10 mo</td>
<td>49%</td>
<td>41%</td>
<td>14%</td>
<td>15%</td>
<td>0</td>
<td>10%</td>
</tr>
<tr>
<td>Brandi et al, 1995</td>
<td>36%</td>
<td>15 mo</td>
<td>26%</td>
<td>74%</td>
<td>12%</td>
<td>15%</td>
<td>0</td>
<td>10%</td>
</tr>
<tr>
<td>Now et al, 1998</td>
<td>98%</td>
<td>12 mo</td>
<td>25%</td>
<td>62%</td>
<td>29%</td>
<td>23%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>De Franceschet et al, 2003</td>
<td>94%</td>
<td>30 mo</td>
<td>14%</td>
<td>76%</td>
<td>12%</td>
<td>7%</td>
<td>2%</td>
<td>9%</td>
</tr>
</tbody>
</table>

Continued results for atherosclerosis and fibrovascular dysplasia cases in the study.

Angioplasty

- RVH 2° Atherosclerosis: Outcomes
  - Poorer outcomes with higher M&M than FD
  - Lower technical success and higher restenosis rates for ostial lesions (up to 35%)

<table>
<thead>
<tr>
<th>Study</th>
<th>No. of Patients</th>
<th>Follow-up</th>
<th>Cured (%)</th>
<th>Improved (%)</th>
<th>Failed (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kim et al, 1985</td>
<td>51</td>
<td>16 mo</td>
<td>27 (unilateral)</td>
<td>30</td>
<td>53</td>
</tr>
<tr>
<td>Lentinger et al, 1989</td>
<td>75</td>
<td>24 mo</td>
<td>23</td>
<td>23</td>
<td>10 (unilateral)</td>
</tr>
<tr>
<td>Lentinger et al, 1993</td>
<td>77</td>
<td>16 mo</td>
<td>25</td>
<td>50</td>
<td>35</td>
</tr>
<tr>
<td>Lentinger et al, 1995</td>
<td>55</td>
<td>13 mo</td>
<td>22</td>
<td>50</td>
<td>35</td>
</tr>
<tr>
<td>Lentinger et al, 1999</td>
<td>130</td>
<td>22 mo</td>
<td>20</td>
<td>22</td>
<td>30</td>
</tr>
<tr>
<td>Lentinger et al, 2001</td>
<td>144</td>
<td>9 mo</td>
<td>37</td>
<td>51</td>
<td>30</td>
</tr>
<tr>
<td>Lentinger et al, 2003</td>
<td>151</td>
<td>8 mo</td>
<td>37</td>
<td>50</td>
<td>30</td>
</tr>
<tr>
<td>Lentinger et al, 2005</td>
<td>147</td>
<td>16 mo</td>
<td>26</td>
<td>36</td>
<td>43</td>
</tr>
<tr>
<td>Lentinger et al, 2007</td>
<td>190</td>
<td>12 mo</td>
<td>20</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>Lentinger et al, 2009</td>
<td>190</td>
<td>13 mo</td>
<td>24</td>
<td>24</td>
<td>10</td>
</tr>
<tr>
<td>Lentinger et al, 2011</td>
<td>190</td>
<td>12 mo</td>
<td>25</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>Lentinger et al, 2013</td>
<td>190</td>
<td>13 mo</td>
<td>23</td>
<td>33</td>
<td>33</td>
</tr>
<tr>
<td>Lentinger et al, 2015</td>
<td>190</td>
<td>13 mo</td>
<td>26</td>
<td>36</td>
<td>36</td>
</tr>
<tr>
<td>Lentinger et al, 2017</td>
<td>190</td>
<td>13 mo</td>
<td>25</td>
<td>33</td>
<td>33</td>
</tr>
<tr>
<td>Lentinger et al, 2019</td>
<td>190</td>
<td>13 mo</td>
<td>25</td>
<td>33</td>
<td>33</td>
</tr>
</tbody>
</table>

Continued with improved results as "benefit" results.
Continued with improved results as "benefit" results.
Angioplasty

- Ischemic Nephropathy & ASO: Outcomes

<table>
<thead>
<tr>
<th>Study</th>
<th>Follow-up</th>
<th>Improved (%)</th>
<th>Stable (%)</th>
<th>Worse (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Martin et al, 1988*</td>
<td>16 mo</td>
<td>43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hayes et al, 1988</td>
<td>13 mo</td>
<td>26(^1)</td>
<td>74</td>
<td></td>
</tr>
<tr>
<td>Jensen et al, 1995</td>
<td>12 mo</td>
<td>74(^1)</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>Karagiannis et al, 1995</td>
<td>39 mo</td>
<td>15</td>
<td>33</td>
<td>52</td>
</tr>
<tr>
<td>Zuccala et al, 1996</td>
<td>37 mo</td>
<td>50</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Paulsen et al, 1999</td>
<td>104 mo</td>
<td>56</td>
<td>21</td>
<td></td>
</tr>
</tbody>
</table>

*Study population with impaired renal function (creatinine >1.7 mg/dL), no report of stable renal function.
\(^1\)Clinical benefit: cured or improved blood pressure or renal function.
\(^4\)Combined with improved results as “benefit” result.

Endovascular Stenting

- Adjunct to angioplasty
- ASO (especially ostium) = main use
- Indications
  - Poor immediate PTA results
  - Restenosis after PTA
  - Artery dissection
  - Intimal flaps
- High technical success rates: 95-100%
- Restenosis rates = 15-20% (intimal hyperplastic rxn)
## Endovascular Stenting

### Table 36-11. Renal Artery Stents: Blood Pressure Results

<table>
<thead>
<tr>
<th>Study</th>
<th>Cured (%)</th>
<th>Improved (%)</th>
<th>Same (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coggins et al., 1994</td>
<td>74</td>
<td>86</td>
<td>0</td>
</tr>
<tr>
<td>Guglielmi et al., 1994</td>
<td>6</td>
<td>45</td>
<td>49</td>
</tr>
<tr>
<td>Lusby et al., 1995</td>
<td>0</td>
<td>44</td>
<td>56</td>
</tr>
<tr>
<td>de Vries et al., 1995</td>
<td>0</td>
<td>75</td>
<td>25</td>
</tr>
<tr>
<td>Lemm et al., 1996</td>
<td>4</td>
<td>35</td>
<td>61</td>
</tr>
<tr>
<td>Hans et al., 1996</td>
<td>4</td>
<td>35</td>
<td>61</td>
</tr>
<tr>
<td>Khan et al., 1997</td>
<td>16</td>
<td>62</td>
<td>22</td>
</tr>
<tr>
<td>Vassilopoulos et al., 1997</td>
<td>6</td>
<td>61</td>
<td>33</td>
</tr>
<tr>
<td>Weiss et al., 1996</td>
<td>1</td>
<td>42</td>
<td>57</td>
</tr>
<tr>
<td>Szatmary et al., 1988</td>
<td>0</td>
<td>55</td>
<td>45</td>
</tr>
<tr>
<td>Glick et al., 1988</td>
<td>0</td>
<td>53</td>
<td>47</td>
</tr>
<tr>
<td>Rodriguez-Lopez et al., 1999</td>
<td>13</td>
<td>55</td>
<td>32</td>
</tr>
</tbody>
</table>

5% 55% 40%

**Combined “clinical benefit” rate.**

### Table 36-12. Renal Artery Stents: Renal Function Results

<table>
<thead>
<tr>
<th>Study</th>
<th>Improved (%)</th>
<th>Stable (%)</th>
<th>Worse (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bier et al., 1994</td>
<td>37</td>
<td>37</td>
<td>26</td>
</tr>
<tr>
<td>Lusby et al., 1996</td>
<td>36</td>
<td>48</td>
<td>18</td>
</tr>
<tr>
<td>White et al., 1997</td>
<td>20</td>
<td>75</td>
<td>5</td>
</tr>
<tr>
<td>Harden et al., 1997</td>
<td>34</td>
<td>24</td>
<td>32</td>
</tr>
<tr>
<td>Dennis et al., 1998</td>
<td>76*</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Tuttle et al., 1998</td>
<td>13</td>
<td>76</td>
<td>11</td>
</tr>
<tr>
<td>Frel et al., 1998</td>
<td>33</td>
<td>62</td>
<td>5</td>
</tr>
<tr>
<td>Kudacki et al., 1999</td>
<td>29</td>
<td>71</td>
<td>0</td>
</tr>
<tr>
<td>Rodriguez-Lopez et al., 1999</td>
<td>55*</td>
<td>45</td>
<td></td>
</tr>
<tr>
<td>Burket et al., 2000</td>
<td>43</td>
<td>24</td>
<td>33</td>
</tr>
<tr>
<td>Perkins et al., 2001</td>
<td>8</td>
<td>57</td>
<td>35</td>
</tr>
<tr>
<td>Szamurthy et al., 2004</td>
<td>87*</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Zeller et al., 2004</td>
<td>34</td>
<td>39</td>
<td>27</td>
</tr>
</tbody>
</table>

25% 60% 15%

*Combined “clinical benefit” rate.
RAS in Children

- Etiologies:
  - Arteritis (Takayasu’s)
  - FD
  - Middle aortic syndrome
  - Neurofibromatosis

- Presentation = RVH

- Mainstay of treatment = angioplasty
  - High initial and retreatment success rates
Renal Artery Aneurysms

- Def’n = localized dil’n of main renal a. or branches d/t weakness in elastic tissue of media

- Incidence = 0.09-0.3%

Classification (4 types)

- Saccular = 75%
  - Often involves branches
  - 25% bilateral or multiple
  - Secondary atherosclerosis & intramural calcification
    (ringlike calcification)
  - Complications:
    - Spontaneous rupture
    - Erosion into renal vein or renal pelvis
    - Mural thrombus & embolization

Campbell’s 9th Ed.
Renal Artery Aneurysms

- **Classification (4 types)**
  - Saccular = 75%
  - Fusiform
    - Young pts with HTN & stenosing FD
    - Uniform dilation (3-4x) beyond stenosis
    - 1-3cm long
    - Don’t calcify
    - Main Cx = thrombosis (low flow from prox stenosis)
  - Dissecting
    - Intimal tear → luminal occlusion
    - Cx = thrombosis & renal infarct; rupture
    - Occur in setting of: ASO, Intimal f., Perimedial f., extension of dissecting AAA
Renal Artery Aneurysms

Classification (4 types)
- Saccular = 75%
- Fusiform = 20%
- Dissecting
- Intrarenal = 17%
  - Congenital, post-traumatic, iatrogenic, neoplastic, PAN
  - Saccular or fusiform; may be calcified
  - Propensity for rupture!!!

Campbell's 9th Ed.

Renal Artery Aneurysms

Signs & Symptoms
- Usually asymptomatic
- HTN (15-75%)
- Flank pain
- Hematuria
- Abdominal bruit
- Palpable pulsatile mass (rare)
Renal Artery Aneurysms

- Complications
  - Peripheral dissection
  - Thrombosis → renal infarction
  - Mural thrombus → emboli
  - Obstructive uropathy
  - Erosion → AV fistula
  - Spontaneous rupture

Renal Artery Aneurysms

- Natural History (small aneurysms)
  - Henriksson et al, 1985
  - 34 RAA’s in 21 patients, serial angiography
  - Mean f/u = 35 months (1-172)
  - Mean age at Dx = 49 yrs; 16F, 5M
  - 12% were calcified; Mean size = 12mm (5-24)
  - 2nd angiogram (mean 24mos) → 88% unchanged (remainder minimal increase size)
  - No ruptures throughout f/u
  - Asymptomatic throughout (3 HTN with 1 RVH)

Henriksson et al, Eur Urol 1985, 11:244
Renal Artery Aneurysms

Who to observe:
- <2cm well-calcified in asymptomatic normotensive pt
- <2cm incompletely (or non) calcified in asymptomatic pt with no indications for surgery:
  - Renal ischemia & HTN
  - Functionally significant RAS
  - Dissecting aneurysm
  - Woman of childbearing age likely to conceive
  - Radiographic evidence of expansion on serial imaging
  - Thrombus on angio with evidence of emboliz’n
  - Symptoms
Renal Artery Aneurysms

- >2cm asymptomatic pt
  - No clear guidelines
  - ?operate on all (Henke, Seki)
  - Intrarenal & incompletely calcified → should probably operate

Campbell's 9th Ed.
Koyanagi et al, Uro Int 2002, 68:24
Seki et al, JU 1997, 158:357

Renal Artery Aneurysms

- Treatment Options
  - Open surgical repair: 96-97% success rates
  - Endovascular (case reports)
    - Embolization (coiling)
    - Stenting (exclude aneurysm)
      - Schneidereit et al, J Endovasc Ther 2003, 10:71-74 – case report of ruptured RAA

Campbell's 9th; Henke et al, 2001
Renal Artery Aneurysms

- Treatment Options
  - Laparoscopic???

LAPAROSCOPIC REPAIR OF RENAL ARTERY ANEURYSM

INDERBI R. GILL*, DAVID P. MURPHY, THOMAS H. S. HSU, AMR FERGANY, HAZEM EL FETTOUH AND ANoop M. MERaney

From the Section of Laparoscopic and Minimally Invasive Surgery, Urological Institute, Cleveland Clinic Foundation, Cleveland, Ohio

- At bifurcation
- Bulldogs, intracorporeal suturing
- WIT = 31 min!!!

Gill et al, JU 2003, 166:202

Renal Artery Aneurysms

- Case#1 conclusion = iatrogenic aneurysm
  - Unstable pt. POD#7 AAA repair
  - Suprarenal clamping; fogarty balloon for renal artery clot
Arteriovenous Fistulas

- Incidence – estimated at <0.04%
  - 200-300 reported cases in literature
  - Case reports & small case series

Crotty et al, JU 1993, 150:1355

Arteriovenous Fistula

- 3 categories:
  - Congenital (22-25%)
    - Supplied by renal a. branch of normal calibre
    - Cirsoi or angiomatous configuration
    - Multiple small interconnecting AV channels
    - Located in lamina propria beneath urothelium
    - Impaired distal renal parenchymal vascularity
    - Early filling of renal vein
    - M=F
    - Present in adult life

Crotty et al, JU 1993, 150:1355
Campbell’s 9th Ed.
Arteriovenous Fistula

- 3 categories:
  - Congenital (22-25%)
  - Idiopathic (3-5%)
    - Single
    - Not cirsoid
    - Angiographically similar to acquired (cause unknown)
    - From erosion of preexisting aneurysm into vein

Crotty et al, JU 1993, 150:1355
Campbell's 9th Ed.

Arteriovenous Fistula

- 3 categories:
  - Congenital (22-25%)
  - Idiopathic (3-5%)
  - Acquired (70-75%)
    - Solitary communication b/w artery & vein
    - Causes:
      - Renal Bx = #1 (4-16% incidence; Maldonado et al 48%!)
      - RCC, renal trauma, inflammation, renal surgery (PNL, PxNx, nephrectomy)

AUAUS 1997, V.16(24)
Crotty et al, JU 1993, 150:1355
Campbell's 9th Ed.
Arteriovenous Fistula

- **Signs & Symptoms**
  - Abdominal bruit (75%)
  - CHF, Cardiomegaly & Diastolic HTN (50%)
  - Hematuria (33%) → 72% congenital AVM’s
  - Tachycardia
  - Palpable flank mass (spontaneous rupture)
  - Allograft dysfunction

<table>
<thead>
<tr>
<th>Signs/Symptoms</th>
<th>% Cenoid (54 cases)</th>
<th>% Anomalous (54 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal bruit</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Hypertension: Systolic</td>
<td>22</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>53</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>7</td>
<td>55</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>Gross hematuria</td>
<td>3</td>
<td>37</td>
</tr>
</tbody>
</table>

Crotty et al, JU 1993, 150:1355

Arteriovenous Fistula

- **Indications for treatment:**
  - HTN
  - CHF
  - Severe hematuria
  - Expanding lesion on serial angiography
  - Rupture
  - Progressive renal failure
  - ?Progressive allograft dysfunction

Kitajima et al, Trans Proc 2000, 32:1911
Arteriovenous Fistula

- Up to 9% will require intervention

- Management – depends on etiology:
  - RCC = immediate Rad Nx
  - Needle Bx = expectant
    - 70% close in 18 mos (native & transplant)
  - Trauma = often expectant

AUAUS 2003, V.22 (39)
AUAUS 1997, V.16(24)
Campbell's 9th Ed.

Arteriovenous Fistula

- Treatment Options:
  - Goal = maximum preservation of renal f'n
  - Embolization (coil & glue)
    - 70-95% technical success using ‘superselective techniques’
      (including post-Bx allograft AVF’s)
  - Surgical
    - Congenital AVM = partial or total Nx
    - Idiopathic/Acquired AVF = as above or identification and
      obliteration of connecting vessel
    - Resolution of HTN: Congenital = 62%; Acquired = 85%

AUAUS 2003, V.22 (39)
Crotty et al, JU 1993, 150:1355
Campbell's 9th Ed.
Arteriovenous Fistula

Case#2 conclusion = congenital AVM
- Healthy 45yo woman c intractable hematuria
  NYD
Thank-you