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Renal Medicine

physiology, transplantation, vascular disease

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Renal Physiology

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Topics to cover

- Proteinuria: diagnosis, guidelines, treatment
- Metabolic urgencies and emergencies
- Myoglobinuria and diabetes insipidus
- Complications associated with transfusion

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Proteinuria

- Etiology
  - Larger molecules transiting from blood to urine through a “leaky” glomerulus
- Diagnosis
  - Dipstick – screening test, predominantly detects albumin
  - Dipstick affected by concentration of urine
  - Further testing with albumin-to-creatinine ratio (ratio reduces variable of hydration), protein-to-creatinine ratio, or 24-hr urine test (gold standard)

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Proteinuria

- Dipstick
  - Trace proteinuria (10-30 mg/dL)
  - 1+ (30 mg/dL)
  - 2+ (100 mg/dL)
  - 3+ (300 mg/dL)
  - 4+ (1000 mg/dL or more)
- >3g per 24-hour = nephrotic range

- Normal values
  - Daily excretion: <150 mg/day
  - Spot albumin to creatinine ratio (ACR): 30 mg/g (3.4 mg/mmol)

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Guidelines

- Lower GFR and increased proteinuria portend worse prognosis

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

GFR categories (ml/min/ 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

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Kidney Int Suppl 3[1]: 2012.

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“Classic” urine findings

- Acute glomerulonephritis: +/- gross hematuria, proteinuria, micro-hematuria, and dysphoric RBCs or red cell casts on urine microscopy
- ATN: bland urine on U/A and muddy brown casts on micro
- AIN: sterile pyuria, +/- white blood cell casts on urine microscopy, rare microhematuria
- IgA, thin basement membrane: isolated microhematuria
- Get a history
  - Meds, SLE, hx of streptococcal disease, unexplained AKI

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AUA guidelines. Renal Mass and Localized Renal Cancer: Evaluation, Management, and Follow-up

- Clinicians should consider referral to nephrology in patients with a high risk of CKD progression, including those with estimated glomerular filtration (eGFR) rate less than 45 mL/min/1.73m^2, confirmed proteinuria, diabetics with pre-existing CKD, or whenever GFR is expected to be less than 30 mL/min/1.73m^2 after intervention. (*Expert Opinion)*

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AUA guidelines. Microhematuria. Initial Evaluation

- Clinicians should refer patients with microhematuria for nephrological evaluation if medical renal disease is suspected. However, risk-based urologic evaluation should still be performed. (*Clinical principle)*

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Proteinuria treatment

- Depends on the quantity and etiology, though typically medical management
- In the U.S., diabetes and HTN account for 2 out of 3 new cases of CKD*
  - BP control
  - Diabetes control
  - RAAS inhibitors (ACE and ARB)
  - Steroids and immunosuppressants

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*cdc.gov/kidneydisease. Accessed 4/14/25.

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Metabolic urgencies and emergencies

- Hyponatremia
  - Excess water relative to sodium (fluid absorption from hypotonic irrigation, SIADH, primary polydipsia)
  - Acute – developed within past 48hrs
  - Headaches, vomiting, disorientation, seizures, coma, brain stem herniation
  - Assess volume status
  - Treat if acute, symptomatic, or severe (120 mEq/L)
    - Free water restriction, normal saline, hypertonic saline, possible loop diuretics, salt tabs
  - Correct slowly (4 to 6 mEq/L per 24hr) to avoid osmotic demyelination syndrome

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Metabolic urgencies and emergencies

- Hypokalemia
  - Causes: GI losses (diarrhea, fistula, NG, vomiting), burns, renal excretion
  - Muscle weakness, cramps, arrhythmias (ST depression, U waves)
  - Treat with KCl (IV and/or oral) and remember to check magnesium
- Hyperkalemia
  - Causes: massive transfusion, GI bleed, hyperglycemia, metabolic acidosis
  - Muscle weakness, arrhythmias (peaked T waves, shortened QT)
  - Treat with IV calcium, IV insulin (10 units regular), IV glucose
  - Dialysis if ESRD patient

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Metabolic urgencies and emergencies

- Hypercalcemia
  - Causes: Ingestion, malignancy, primary hyperPTH, granulomatous disease
  - Sx's: Fatigue, poor concentration, headaches, depression, arrhythmias, GI
  - Tx: NS, loop diuretics, bisphosphonates, parathyroidectomy

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Myoglobinuria

- Stores oxygen, intracellular, skeletal muscle
- Rhabdomyolysis results from muscle necrosis
  - Classic triad – muscle pain, weakness, dark urine (rare)
  - Renal damage: obstruction of tubules, toxicity to tubular cells
- Risk factors: ↑OR time, ↑BMI, male gender
- Labs: CK 5x norm, hyperkalemia, AKI, + heme dip and - RBC micro
- Tx: early & aggressive IV crystalloids, +/- bicarb-> ↑urine pH >6.5

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Diabetes insipidus

- Arginine vasopressin = vasopressin = ADH
  - Desmopressin (synthetic) = DDAVP (vasodilatory shock, certain forms vWD)
  - Sodium must be monitored closely
- Arginine vasopressin deficiency (central DI)
  - Polyuria (3L/day), nocturia, polydipsia
  - Tx - desmopressin
- Arginine vasopressin resistance (nephrogenic DI)
  - Polyuria, nocturia, polydipsia
  - Tx – thiazide diuretics, low salt diet, low/normal protein diet

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Complications associated with transfusions

- Allergic reaction – itching, rash, breathing difficulty
- Transfusion-related acute lung injury (TRALI) – 1 in 5,000
  - Hypoxemia, pulmonary infiltrates, fever, hypotension (within 6 hours)
- Infection – 1 in 300,000 (Hep B)*
- Volume overload, hyperkalemia, iron overload
  - typically associated with large volume or repeated transfusions
- Immunologic – hemolytic reaction, sensitization

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*redcrossblood.org. Accessed 4/15/23.

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Renal Transplantation

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Topics to cover

- Evaluation of living donors
- Urologic considerations
- Surgical complications of renal transplant

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Living kidney donation

- Approximately 75% of interested kidney donors have a condition that makes donation risky or inadvisable
- Open donor nephrectomy is rare in United States
- Majority are left sided due to increased vessel length
- Leave the donor with the “better” kidney

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Who makes a good kidney donor?

- >18 years old
- GFR >80
- Reliable access to health care, no coercion
- BMI <35
- Non-smoker
- “Low risk” of developing CKD
  - Medical history, family history, age, medications

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Risks with living donation

- Small increased risk of ESRD (0.5 events per 1000 per years) and preeclampsia
- Slight rise in diastolic BP
- No significant change in life expectancy
- Mortality 0.01% (90 day)
- Any complication 10-15%
  - Major (Clavien IV or higher) 2.5%

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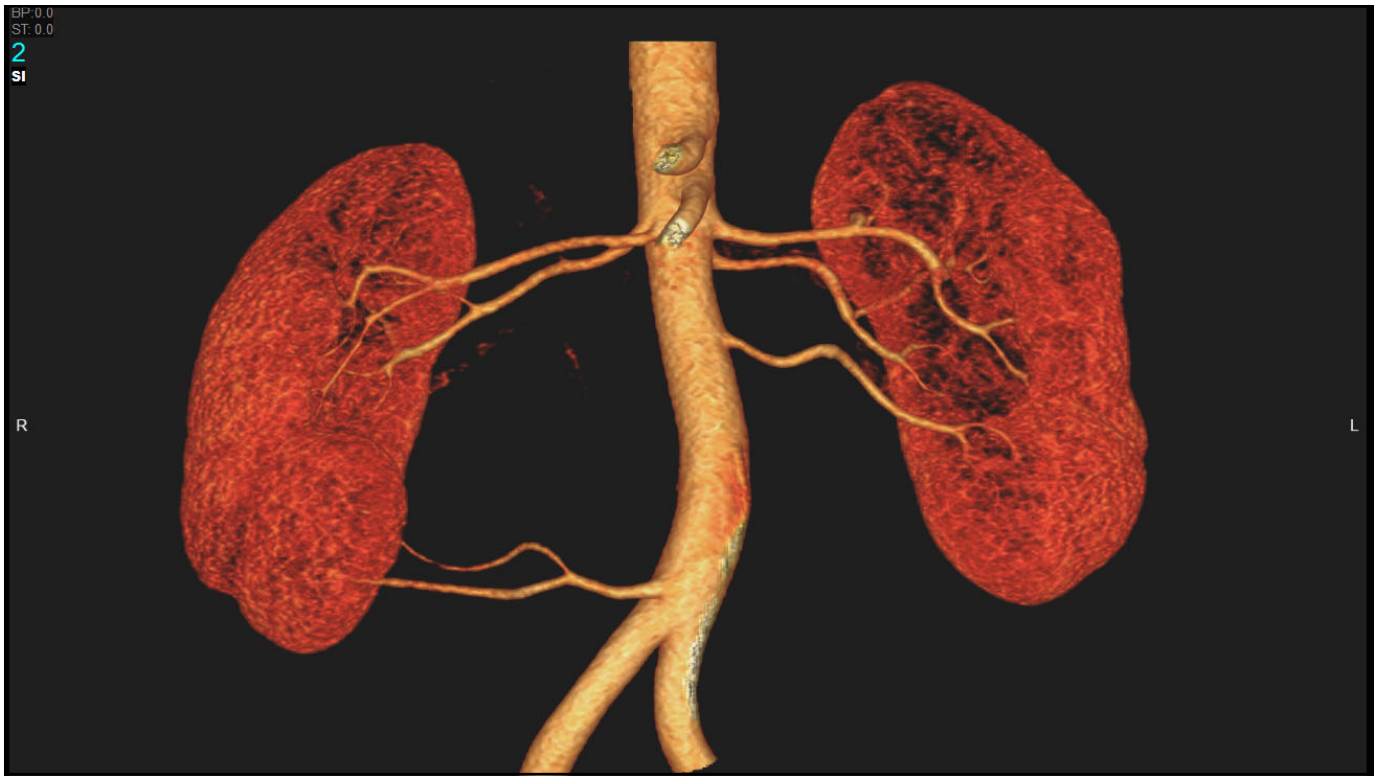

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Urologic considerations in renal transplant

- Lower urinary tract
  - CIC safe and effective
  - Transplant to augment or diversion similar long-term outcome
  - “Dry TURP" prone to strictures and contractures
  - Bladders will expand unless fibrotic
- Pelvic anatomy
  - Vessels with minimal atherosclerosis, no significant stenoses
  - Prior radiation, node dissection, augmentation
- GU malignancy
  - Determine risks of recurrence, potential medical/surgical therapies

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Surgical risks

- Urine leak or obstruction/stricture (4-5%)
  - may need re-operation -> low risk to lose kidney
  - endoscopic approaches
  - surgery for early high-volume leaks, necrotic ureter or endo failure
- Vascular leak, dissection, thrombosis (1-2%)
  - re-operation, endovascular repair -> high risk to lose kidney
- Lymphocele (5-10%)
  - percutaneous drainage, sclerosis, peritoneal window
- Wound infection/ hernia

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Management of early graft dysfunction

- What was the anticipated trajectory of the allograft?
- Ddx: ATN, acute rejection, drug toxicity, urinary obstruction vascular compromise
  - (dissection, thrombosis, renal artery stenosis)
- Check Foley, bladder scan
- Fluid bolus, diuretics, drug levels
- Duplex ultrasound, biopsy

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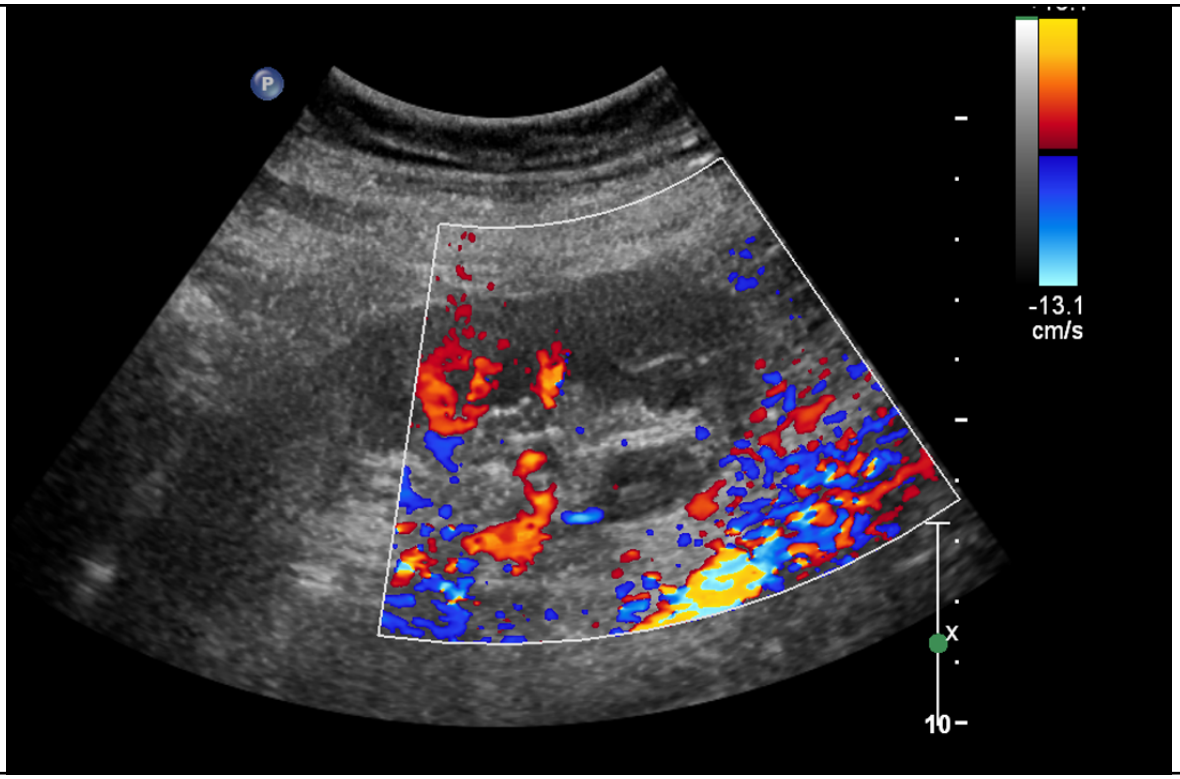

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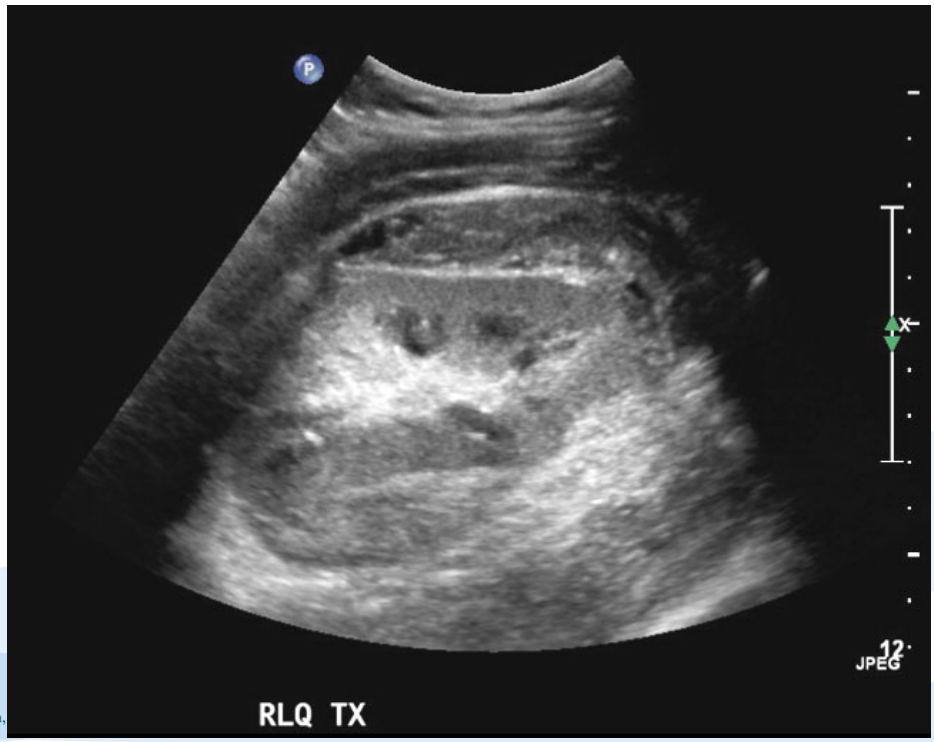

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Renovascular Disease

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Overview

- Pathophysiology
- Classification of lesions
- Clinical evaluation
- Medical and surgical management
- Renal artery aneurysm

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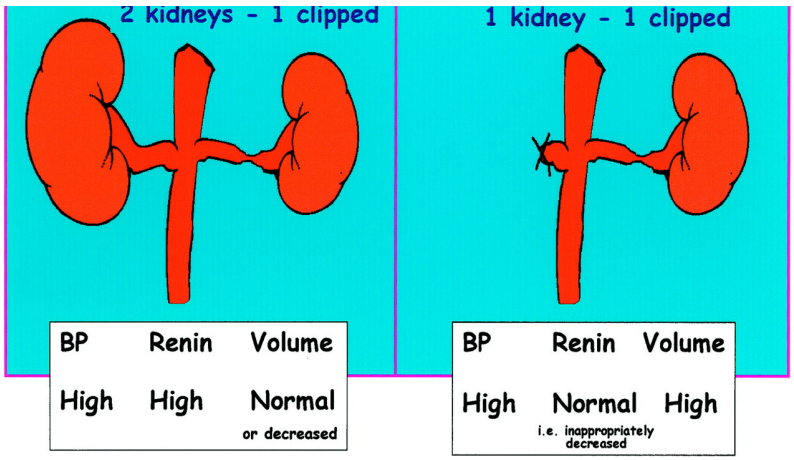
Goldblatt dog models



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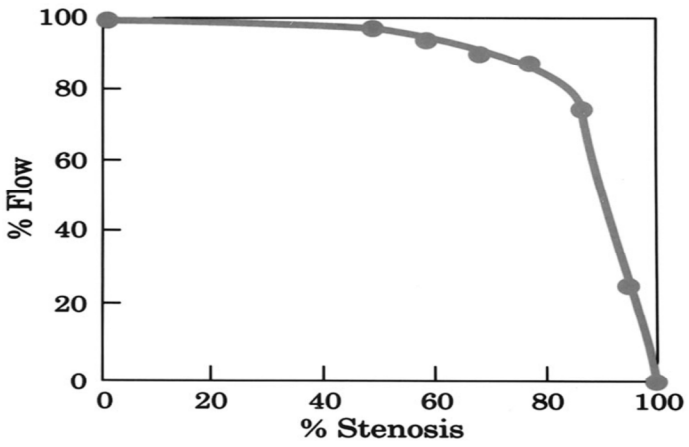
Diameter and blood flow



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Classification

- Atherosclerotic ~90%
- Fibrous dysplasia ~10%
- Vasculitis, congenital bands, extrinsic compression (neoplasia/radiation)

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Atherosclerotic disease

- Typically, a systemic disease
- Involves proximal 2cm of artery
- Progression common, at least 50% in 2 years
- 10-15% progress to occlusion
- Can cause HTN and renal failure

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Fibromuscular dysplasias

- Medial fibroplasia 80-90%
- Intimal fibroplasia 10%
- Perimedial fibroplasia rare
- Fibromuscular hyperplasia rare

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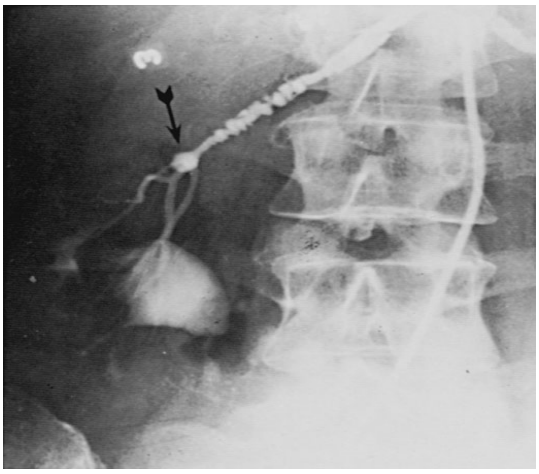
Medial fibroplasia

- Most common
- Females 20-60
- Commonly bilateral
- “string of beads”
- Involves distal 2/3rd and branches
- Progression less common



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Clinical clues to renovascular HTN

- HTN onset <30 yrs
- Sudden or accelerated onset
- Severe HTN
- Lack of family history
- Difficult to control
- Bruits
- GFR decline with BP reduction or after initiation of ACE/ARB
- Renal size disparity

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Key diagnostic points

- Duplex ultrasound
  - Widely available, low-cost, serial exams, operator dependent, body habitus
- MRA and CTA
  - Excellent anatomic detail, no physiologic data, contrast in CKD
- Renal angiography
  - Gold standard, invasive
- Radionuclide renography
  - Captopril provocation, largely fallen out of favor
- Renal vein renin sampling
  - Invasive, stringent testing conditions

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Investigation of renovascular HTN

- High suspicion
  - Confirm with duplex U/S
  - Angiography
- Mild to moderate suspicion
  - Non-invasive imaging
  - If significant azotemia, U/S rather than MRA or CT

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Treatment of renovascular HTN

- Multiple randomized trials have failed to show significant benefit of renal artery stenting in majority of patients
- Medical management remains the mainstay of treatment
  - Medial fibroplasia and atherosclerotic (without ischemia nephropathy) best for medical
- Angioplasty +/- stent can still be an option in select cases
- Rarely, nephrectomy if small and non-functioning

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Renal artery aneurysms

- Most small and asymptomatic
- Pathology
  - Saccular (70-90%), fusiform, dissecting, intrarenal
- Risk factors for rupture
  - Absent/incomplete calcification, >2cm diameter, expanding, HTN, pregnancy
- Complications
  - Retroperitoneal bleeding, pain, hematuria
- Treatment- endovascular primarily

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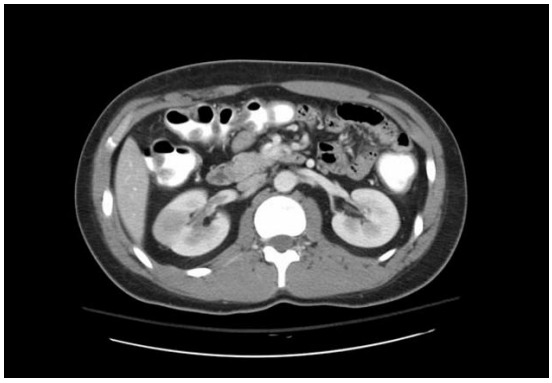
Renal arterial infarct



- Thrombotic, embolic, or dissection
- Atrial fibrillation, hypercoag states risks
- Acute flank pain, mimics stone or pyelo
- May have hematuria, elevated LDH
- Missed on CT KUB unless contrast
- Renal scan most sensitive
- Depending on clinical scenario, anticoagulation and endovascular intervention most common

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Question

A 32-year male is postoperative day 4 from an RPLND. He has an ileus and high output NG tube volumes. He is noted to have mildly symptomatic hypokalemia. In addition to checking an EKG and replacing potassium, the following lab that should also be checked is:

A. Calcium.
B. ACTH.
C. Sodium.
D. Magnesium.
E. Cortisol.

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Question

Injury to this commonly encountered nerve during mobilization of the iliac vessels for renal transplantation will result in:

A. difficulty with thigh adduction.
B. numbness of the upper medial thigh.
C. difficulty with hip flexion and numbness the anterior thigh.
D. numbness of the anterior one-third of the scrotum and base of penis.
E. numbness of the posterolateral gluteal skin and suprapubic area.

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Question

A healthy 25-year-old female is diagnosed with new onset severe HTN after presenting with headaches. They are started on an ACE inhibitor with a subsequent rise in their creatinine from 0.8 to 1.6 mg/dl. The most likely cause of their hypertension is:

A. Medial fibroplasia
B. Intimal fibroplasia
C. Perimedial fibroplasia
D. Fibromuscular hyperplasia
E. Renal artery aneurysm

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