Examination of the Urine is part of the Nephrologists physical exam

Dr. M.T. Michaud
Nephrologist
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Kidney Disease

- Patients presenting with kidney disease can be very symptomatic
  - Gross hematuria
  - Hypertension
  - Flank pain
  - Edema
  - Uremia
- But they can also be Completely asymptomatic and identified on routine labs
  - abnormal urinalysis
  - elevated Cr and urea
Kidney disease

• When we suspect kidney disease we order a Cr and urea. These labs tell us the severity of the kidney disease but not the etiology

• To identify the etiology we need:
  – a urinalysis with a dip and a microscopy
  – diagnostic imaging – an US, sometimes a renal scan or a CT
  – sometimes a renal biopsy
Urinalysis

• Urine colour
  – Normal is clear to light yellow, lighter when dilute, darker when concentrated
  – Red to brown
    • Supernatant clear after centrifuge
      – Hematuria
    • Supernatant red/brown after centrifuge
      – Hemoglobinuria, myoglobinuria
      – Certain drugs, beets, porphyria, food dyes
      – Semen
      – Alkaline urine
  – False positive dips for hematuria are common, false negatives are rare
Urinalysis

• Dipsticks also detect
  – Leukocyte esterase (pyuria)
    • UTI/Pylonephritis
    • Acute interstitial nephritis
    • Nephrolithiasis
    • Renal TB
  – Nitrites
    • Enterobacteriaceae
      – E.coli, salmonella, shigella, proteus enterobacter, serratia, citrobacter, klebsiella, yersinia
Urinalysis

• Urine dipstick
  – Detects albuminuria
    • Falsely low in dilute urine
    • Falsely high in concentrated urine
    • False positive after IV contrast
    • Elevated when febrile and after exercise
  – Dipstick does not measure other proteins such as light chains
  – Only positive when proteinuria is $> 300$ mg/day
  – Normal is $< 30$ mg/day
  – $30 – 300$ mg/day is microalbuminuria
    • Earliest clinical manifestation of diabetic nephropathy
    • Marker of increased cardiovascular risk in nondiabetics as well as diabetics
Urinalysis

• Quantitation of urine protein
  – Urine dip inaccurate
  – 24 hour urine
  – Protein/Cr ratio

• Specific gravity
  – < 0.003 dilute, > 0.03 concentrated

• Glucose
  – Glucosuria does not occur until plasma glucose is > 10 or there is proximal tubular dysfunction
Urine microscopy

• Tamm Horsefall mucoprotein is secreted by the epithelial cells of the loop of henle, distal tubules, and collecting duct

• The Tamm Horsefall mucoprotein is the base material of all casts in the urine, the glue, and is the most common protein in normal urine
Hyaline Cast

- Composed of only Tamm-Horsfall mucoprotein
- More common in volume depleted patients
Acute Tubular Necrosis (ATN)

- Ischemic or toxic injury to the tubules causes injury to the renal tubular epithelial cells.
- Sepsis, MI, IV contrast, severe volume depletion, hypotension
- When injured epithelial cells die they slough off and are caught up in developing hyaline casts. They form the 3 types of casts of ATN
  - Cellular casts
  - Coarsely granular casts
  - Finely granular casts
Cellular casts of ATN (Early on in ATN)
Finely Granular Casts (Epithelial cells have degenerated to the point of being unrecognizable)
White cell cast

-Pylonephritis
-Acute interstitial nephritis
-Genitourinary TB
-Renal lithiasis
Hematuria

• Can originate from any site in the urinary tract or the vagina
  • Glomerular
    – Glomerulonephritis
  • Extraglomerular (Urologic or gynologic)
    – Inflammation
    – Infection
    – Invasion/cancer
    – Stones
Non-gglomerular hematuria
Non-glomerular hematuria

- R/O infection
- Assess for malignancy
  - Smoking
  - Occupational dyes
  - Age > 40 yrs
  - Frequent analgesic use
- Stones
Glomerular hematuria = Dysmorphic RBC
Dysmorphic RBC
Variations in size, shape and hemoglobin content
• Phase contrast Microscopy
Red Cell Cast
Glomerular hematuria

• Nephritic or nephrotic syndrome

• Nephritic
  – Htn
  – Rapidly progressive renal failure
  – Non-nephrotic range proteinuria (< 3.5 gm/day)
  – Active urinary sediment
Nephritic syndrome

- Postinfectious
- MPGN
- Lupus
- IgA
- Anca-associated
- Goodpastures syndrome
Nephrotic syndrome

- Proteinuria > 3.5 g/day
- Hypercholesterolemia
- Edema
- Hypoalbuminemia
- Lipiduria
- Less active urinary Sediment
- Thrombotic disease
Nephrotic syndrome

- FSGS
- Minimal change disease
- Membranous Nephropathy
- Amyloidosis
- Diabetic nephropathy
- Postinfectious
- Pre-eclampsia
Case 1

51 yo female
- 2 weeks progressive cough, dyspnea, decreased oral intake
- several months of malaise, weight loss, fatigue, arthralgias
- chronic sinusitis
- Admitted to ICU with hypotension, respiratory failure and acute kidney injury
Case 1

• Differential diagnosis
  – ?Pneumonia with ATN?
    • Antibiotics
    • Fluid resuscitation
  – ?Vasculitis?
    • High dose steroids
    • Cyclophosphamide
Case 1

• Urine R and M
  – Dysmorphic RBC
  – RBC casts
  – Granular casts

• Urine Protein/Cr ratio
  – 250 gms/mol
  – Equivalent to 2.5 gms/d
ANCA-Associated Vasculitis

1) Granulomatosis with polyangiitis (Wegeners)
2) Microscopic polyangiitis
3) Churg Straus
   – Common in our area
   – All are associated with anti-neutrophil cytoplasmic antibodies
   – Fever, migratory arthralgias, sinusitis, nose bleeds, cough, dyspnea, fatigue, weight loss, otitis media, nasal crusting, hearing loss
ANCA-Associated Vasculitis

- Pauci-immune
- Dysmorphic hematuria, red cell casts, proteinuria
ANCA-Associated Vasculitis

• Treatment
  – Plasmapheresis
  – High dose steroids
  – Cyclophosphamide

• Followup
  – Monthly Cr, urea, CBC, urine R and M, urine protein/Cr ratio
Case 2

• Found with decreased LOC in the hot Kamloops sun after overdosing on opioids
• Body weight was resting on her right calf
• Hypotensive and volume depleted with a tensely swollen right leg
Case 2

- Progressively increasing Cr
- Ck > 33,000
- Urine coca cola coloured
- Dipped positive for blood
- Microscopy negative for RBC
“Muddy brown” casts
Granular casts in the biopsy
Case 2 - Rhabdomyalysis

- Faciotomy
- Alkalinization of Urine
- High dose IV fluids
Compartment Syndrome

- Life and limb threatening
- Compression of nerves, blood vessels and muscle in a closed space
- Death of tissue, elevated Ck
- Most often involves the forearm and lower leg
Case 3

• 48 yo female rancher
• Right flank pain x 3 hours – worse then labour
• Associated with N/V, decreased oral intake
• Past medical history
  – G2P2
  – Crohn’s disease
  – Not currently on any medications
• Cr increased to 200 umol/L
Urine microscopy
Renal Lithiasis

- Renal calculi affect 5% of Canadian men and women during their lifetimes.
- Most stones result from a complex interaction between diet, fluid, genetic predisposition.
Renal lithiasis

• Risk factors for stones
  – Volume depletion (supersaturation of stone constituents)
  – High salt intake (causes hypercalciuria)
  – Male sex (average first stone at age 30)
  – High meat intake (creates acidic urine, depletes citrate, promotes uricosurea)

• Treatment
  – Calculi smaller than 5mm usually pass spontaneously
  – Once stone passed 24 hour urine to assess for metabolic abnormality
    • Ca, oxalate, uric acid, citrate, mg, Cr, Na, PO4, pH, volume
Calcium Oxalate
Case 3

- 21 yo student
- Sore throat 1 to 4 weeks prior to presentation
- saw GP, unwell, nonspecific symptoms
- Cr 540
- Elevated ASO
- Low complement
- Urine dysmorphic RBC and 1 gm/day proteinuria
Post-strep GN

- Also occurs post staph and less commonly with other bacteria
- Acute immune complex GN
- Good prognosis in children
Case 4

• 43 yo women
  – Large PE
  – Large residual DVT
  – Bilateral pedal edema
  – HTN
  – Anemia and possible GIB

• Labs showed normal Cr and electrolytes, low hgb at 82 and proteinuria of 15g/d with dysmorphic hematuria
PE
IVC Filter

25 mm
Renal biopsy
Treatment

- LMWH/coumadin for hypercoagulable state
- ACE I, statin,
- High dose prednisone
- Trial of cyclophosphamide with no response
- Trial of cyclosporine leading to remission
- Relapse with titration down of cyclosporine
- Now on long term cyclosporine
- Monthly urine studies to monitor for repeat relapse
Urine studies

• A crucial part of the nephrologists physical exam
• Detailed description invaluable to making the diagnosis and following patients with kidney disease long term
• Thank you