North Carolina Academy of Physician Assistants

Recertification Exam Review 2017

RENAL DISEASES REVIEW

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DISCLOSURES

• NONE
LEARNING OBJECTIVES

• Acute Kidney Injury (Acute Renal Failure)
• Chronic Kidney disease
• Glomerulonephritis
• Nephrotic Syndrome
• Polycystic Kidney Disease
• Renal Vascular Disease
• Fluid & Electrolyte Disorders
• Hypervolemia/Hypovolemia
• Acid Base Disorders
ACUTE KIDNEY INJURY

3 Types

1. **Prerenal**
   - Decreased renal blood flow, hypovolemia

2. **Intrarenal (Intrinsic)**
   - Direct kidney damage

3. **Postrenal**
   - Obstruction of urine outflow
ACUTE KIDNEY INJURY - PRERENAL

REDUCED RENAL PERFUSION*

- GI losses: vomiting, diarrhea
- Renal losses: diuretics, polyuria
- Blood/fluid loss: hemorrhage, burns, pancreatitis

MC type. May lead to ATN if not corrected.

Hallmarks: FeNa <1%*, BUN/Cr >20:1*, ↑urine spp gravity.

Mgmt: volume repletion (rapid response).
Obstruction to urinary outflow

*Ex: BPH, malignancy*

- May lead to **hydronephrosis**

**Mgmt:** removal of obstruction (rapid response).
ACUTE KIDNEY INJURY - INTRINSIC

- renal parenchymal insult with nephron damage
  ➞ cast formation*

ATN (Acute Tubular Necrosis):
  - Ischemic: prolonged prerenal, hypotension
  - Nephrotoxic: Aminoglycosides, contrast dye

- UA: epithelial cell or muddy brown casts*
  - Low urine specific gravity

- Mgmt: remove offending agents, IV fluids
ACUTE KIDNEY INJURY - INTRINSIC

- renal parenchymal insult with nephron damage
  ⇒ cast formation*

AIN (Acute Interstitial Nephritis)
- Drug HSN: Penicillin, sulfa drugs, autoimmune

- Triad: rash, eosinophilia, fever
  - UA: white blood cell casts*

- Mgmt: remove offending agent(s).
ACUTE KIDNEY INJURY - INTRINSIC

- renal parenchymal insult with nephron damage
  \(\Rightarrow\) cast formation*

AGN (Acute Glomerulonephritis):

- **UA**: red blood cell casts*
- **Mgmt**: supportive
Renal transplant for end stage disease (stage 5).

**Dialysis:** GFR ≤ 10mL/min &/or serum creatinine ≥8mg/dL

Dialysis in diabetics:
  GFR ≤ 15ml/min &/or serum creatinine of ≥6.
CHRONIC KIDNEY DISEASE

ETIOLOGIES

Diabetes Mellitus: MC cause of ESRD*

Hypertension: 2nd MC cause.

Glomerulonephritis, Polycystic Kidney Disease

DIAGNOSIS

1. PROTEINURIA: single best predictor of disease progression.*
   Spot UAlbumin/UCreatinine Ratio or 24h urine collection.

2. URINALYSIS: abnormal sediment: broad waxy casts seen in ESRD

3. Estimated GFR
CHRONIC KIDNEY DISEASE

MANAGEMENT

**Hypertension:** BP goal <140/90 (JNC8)

**Diabetes control:** Hemoglobin A1C <7.0

**Proteinuria:** protein restriction, ACE Inhibitors or ARB*

Correct anemia: Fe replacement, Erythropoeitin
GLOMERULONEPHRITIS

Inflammation of the glomeruli ⇒ **RBC & protein leakage**

**Proteinuria and Hematuria**

- IgA Nephropathy (Berger’s disease)
- Post infectious
- Membranoproliferative/mesangiocapillary

RAPIDLY PROGRESSING GLOMERULONEPHRITIS

- Goodpasture Syndrome
- Vasculitis
GLOMERULONEPHRITIS

CLINICAL MANIFESTATIONS

• Hematuria

• **Edema**: peripheral, periorbital especially in pediatric patients secondary to proteinuria.

• Hypertension

• **Azotemia**: ↑BUN/Cr, Oliguria

• Fevers, abdominal pain, flank pain
GLOMERULONEPHRITIS - IgA Nephropathy

Aka Berger disease
• MC cause of AGN in adults worldwide.
• MC young males within days (24-48h) after URI or GI infection.

Diagnosis: IgA deposition* on renal biopsy.

Mgmt: ACE inhibitors ± Corticosteroids
GLOMERULONEPHRITIS - Post Infectious

- **MC after GABHS** but can occur after any infection.
- Classically: *2-14y boy with puffiness of eyelids, facial edema up to 3 weeks after strep infection with scanty cola-colored (dark) urine.*

**Diagnosis:** Clinical,
Renal biopsy: immune humps IgM, IgG.*

**Mgmt:** ACE inhibitors ± Corticosteroids.
GLOMERULONEPHRITIS - Goodpastures

- anti-GBM antibodies against the kidney and Type IV collagen antibodies of the lungs (develop glomerulonephritis and hemoptysis*)

- Diagnosis: linear IgG deposits*

- Management: high dose steroid immunosuppression + cyclophosphamide* plus plasmapharesis
NEPHROTIC SYNDROME

HALLMARKS
• Proteinuria
• Hypoalbuminemia
• Edema & hyperlipidemia
NEPHROTIC SYNDROME – MINIMAL CHANGE DISEASE

• MC cause of nephrotic syndrome in children.
• ETIOLOGIES: idiopathic ± associated with viral infections, *allergies* (eX NSAIDs), Hodgkin dz, SLE.

DIAGNOSIS
No visible cellular changes seen on simple light microscopy (or minimal changes).

MANAGEMENT:
• *Corticosteroids* excellent prognosis
NEPHROTIC SYNDROME –
FOCAL SEGMENTAL GLOMERULOSCLEROSIS

- Sclerosis (fibrosis) within the glomerulus.
- Idiopathic, **HTN (especially African-Americans)**,* IV heroin abuse, HIV, reflux nephropathy.
NEPHROTIC SYNDROME – MEMBRANOUS

- **Membranous**: due to SLE, viral hepatitis, malaria, drugs (pencillamine) hypocomplementemia. Usually present with **nephritic-nephrotic picture**.

- **Hallmark**: Thickened basement membrane
NEPHROTIC SYNDROME

DIAGNOSIS
1. 24h urine protein collection of >3.5g/d = gold standard*

2. UA: proteinuria, “Maltese cross” fat casts pathognomonic*

MANAGEMENT:
Corticosteroids in Minimal Change Disease*

Edema reduction: diuretics

Proteinuria reduction: ACEI,* ARB’s
Hyperlipidemia reduction: diet modifications & meds
POLYCYSTIC KIDNEY DISEASE

- Autosomal dominant disorder of genes PKD1 or PKD2
- Kidney cysts & other organs (liver, spleen, pancreas).

CLINICAL MANIFESTATIONS
- Pain: flank or abdominal pain, hepatomegaly
- Hematuria: cystic rupture into the renal pelvis results in gross hematuria.
- Nephrolithiasis, decreased urine concentrating ability, microalbuminuria.
POLYCYSTIC KIDNEY DISEASE

DIAGNOSIS

• Renal Ultrasound: *best initial test*

MANAGEMENT

• **Lifestyle changes:** protein restriction, lowered salt intake, decreased caffeine intake, increased daily water intake.

• **Tight Blood pressure control:** to decrease activation of RAAS. *(ACEI/ARBs).* Goal 140/90
RENOVASCULAR HYPERTENSION

• HTN due to renal artery stenosis 1 or both kidneys ⇒ perceived hypotension ⇒ RAAS activation.

• MC cause of secondary HTN!*

ETIOLOGIES

1. Atherosclerosis MC in elderly*

2. Fibromuscular dysplasia
   MC cause in women <50y.*
RENOVASCULAR HYPERTENSION

CLINICAL MANIFESTATIONS:
severe/refractory HTN, Headache
Abdominal (renal) bruit

DIAGNOSIS
• CT or MR angiography, Captopril renography.
• Renal arteriography: gold standard.*
MANAGEMENT

Surgical: revascularization
• Angioplasty with stent – definitive.*

Medical
• ACE inhibitors*/ARBs (inhibits aldosterone & angiotensin II-mediated vasoconstriction.

• However, ACEI/ARB contraindicated if bilateral stenosis or solitary kidney* can markedly reduce renal blood flow & GFR.
HYPERKALEMIA

ETIOLOGIES

📍 Renal excretion: acute or chronic renal failure,* Hypoaldosteronism, adrenal insufficiency

 )[Meds: K⁺ supplements, K⁺ sparing diuretics, ACEI/ARB ’s, β-blockers, digoxin, NSAID's, cyclosporine

📍 Cell lysis: rhabdomyolysis, burns, hypovolemia, thrombocytosis, leukocytosis (intracellular release of K from cell lysis.

📍 K⁺ Redistribution: metabolic acidosis* (DKA), catabolic states

• Pseudohyperkalemia: venipuncture MC, lab error.
HYPERKALEMIA

MANIFESTATIONS

**Neuromuscular**: weakness (progressive ascending), fatigue, paresthesias, paralysis

**Cardiovascular**: palpitations, cardiac arrhythmias
HYPERKALEMIA

DIAGNOSIS

1. Serum K > 5.0 mEq/L

1. **ECG:** *Tall peaked T waves* → QR interval shortening, QRS widening and prolonged PRI → P wave flattening, *sine wave*
HYPERKALEMIA

MANAGEMENT

- **STEP 1: identify cardiac toxicity:**
  K > 6.5 or severe ekg findings, give calcium gluconate to stabilize the myocardium*

- **STEP 2: remove excess intake/meds:**

- **STEP 3: enhance potassium intracellular shift:**
  - insulin + glucose
  - beta agonists (high dose)
HYPOKALEMIA

ETIOLOGIES

- **Increased urinary/GI losses:** 
  - vomiting, diarrhea, diuretic therapy, renal tubular acidosis – distal (I), proximal (II).

- **Increased intracellular shifts:** metabolic alkalosis, increased β2 activity, hypothermia and chloroquine use

- Decreased K intake: very rare

* If hypomagnesemia present, it may be hard to replenish potassium
HYPOKALEMIA

CLINICAL MANIFESTATIONS

- **Neuromuscular:**
  - severe muscle weakness (resp), cramps
  - rhabdomyolysis, myoglobinuria
  - nausea/vomiting, ileus
  - polyuria

- **Cardiovascular:** palpitations, cardiac arrhythmias
HYPOKALEMIA

DIAGNOSIS

- Serum K < 3.5 mEq/L

- ECG: T wave flattening → prominent U wave*
HYPOKALEMIA

MANAGEMENT

Potassium replacement:

- **KCl** oral if possible

- **IV KCl** given for rapid treatment/severe sx

- Replenish Magnesium if hypomagnesemnecic
HYPERNATREMIA

[Na] > 145 meq/L. Not as common as hyponatremia.

Cause is dehydration. Simply, the inability to drink free water (water loss)

Seen in patients who don’t have access to free water (infants, elderly, debilitated patients, nursing home)
**HYPERNATREMIA**

- **CNS dysfunction**
  - Water shifts out of cells → *shrinkage of brain cells*
  - (increased risk of SAH, intracranial hemorrhage).

- Confusion, lethargy, sz (not as common as hyponatremia), coma, muscle weakness. Lethargy

- Symptoms vary with degree and rapidity of hypernatremia. Chronic hypernatremia generally less symptomatic as a result of adaptive mechanisms.
REVIEW OF HYPERNATREMIA

ECF Volume

HYPOvolemic

Renal loss ($U_\text{Na} > 20$)

- Uosm 300-600
  - Severe Hyperglycemia
  - Osmotic Diuretics

Extra-renal loss ($U_\text{Na} < 10$, Uosm < 400)

- Sweating
- Resp Loss
- GI loss (N/V/D)
- Dehydration

ISOvolemic

Uosm < 250

- Diabetes insipidus
- Reset Osmostat

HYPERvolemic

- Hypertonic Saline
- Mineralocorticoid Excess
HYPERNATREMIA

MANAGEMENT

- **Only hypotonic fluids are appropriate** (ex D5W, pure water, 0.45% NS, 0.2% saline)

  Preferred route is PO

- Correction should be ≤0.5mEq/L/h
  
  *(to prevent cerebral edema.)*

- Except in cases of frank circulatory compromise 0.9% NS is unsuitable for managing hypernatremia.
**HYPONATREMIA**

**Hyponatremia** = ↓ Serum [Na] <135 = increased free water (intracellular).

Due to **impaired kidney free water excretion (increased ADH secretion)** kidney unable to make dilute urine.

**Clinically significant hyponatremia is hypotonic hyponatremia.**
REVIEW OF HYponatremia

If pt critical, Hypertonic saline + Loop diuretic

**Serum OSM**

- Low
  - Hypotonic Hyponatremia (TRUE HYponatREMIA)

- Normal
  - Lab Error (Protein, TG)

- High
  - Hyperglycemia
  - Mannitol

*Note: all have ↑ADH
- SIADH: inappropriate
- Rest: appropriate
hypervolemia

Peripheral and presacral edema
Pulmonary edema
Jugular venous distension
Hypertension
Decreased hematocrit
Decreased serum protein
Decreased BUN: creatinine
HYPOVOLEMIA

- poor skin turgor
- dry mucous membranes
- flat neck veins
- hypotension
- increased hematocrit
- increased serum protein
- increased BUN: creatinine ratio >20:1
- UNa <20 mEq/L

ISOVOLEMIA

absence of the signs of hyper or hypervolemia
REVIEW OF HYPONATREMIA

If pt critical, Hypertonic saline + Loop diuretic

Serum OSM

Low

Lab Error (Protein, TG)

High

Hyperglycemia
Mannitol

ECF Volume

Low

Hypotonic Hyponatremia

Normal

• CHF
• Cirrhosis
• Nephrosis

High

Renal loss ($U_{Na} > 20$)
• Diuretics
  • Thiazide
  • K-sparing
• ACE-I, ARB
• IV RTA, Hypoaldo
Mgmt: Normal Saline

Extra-renal loss ($U_{Na} < 10$, $FeNa < 1$)
• Bleeding
• Burns
• GI (N/V, diarrhea)
• Pancreatitis

Mgmt: Water Restriction

• Hypothyroidism
• AI
• SIADH, post op
• Reset Osmostat
• Water Intoxication
  1° Polydipsia

*Note: all have ↑ADH
• SIADH: inappropriate
• Rest: appropriate

Mgmt: H2O/salt restriction
HYPONATREMIA

MANAGEMENT

Acute hyponatremia (<48h) can be safely corrected more rapidly than chronic hyponatremia.

*Rapid correction of hyponatremia can cause osmotic demyelination* (rapid shrinking of brain cells leading to quadriplegia and other neurologic sequelae).

*Correction should be ≤0.5mEq/L/h*  
(1-2mEq/L/h in severe/symptomatic patients)
**HYPOMAGNESEMIA**

- **GI losses:**
  - Malabsorption: ETOHics, * Celiac sprue
  - Small bowel bypass
  - Chronic diarrhea
  - Laxative abuse

- **Renal losses:**
  - *Diuretics*: thiazides, loop diuretics*
  - Diabetics

  - Primary hyperparathyroidism, primary hyperaldosteronism
  - Renal tubular acidosis
  - *Meds*: amphotericin B, cisplatin, cyclosporine, Proton pump inhibitors
HYPOMAGNESEMIA

CLINICAL MANIFESTATIONS

• **Neurovascular**: AMS, lethargy, seizures, weakness, muscle cramps, vertigo, ↑DTR

• **Hypocalcemia**: Trousseau’s/Chvostek’s due to impaired PTH secretion/release

• **Hypokalemia*** (40-60%)

• **Cardiovascular**: arrhythmias, palpitations
HYPOMAGNESEMIA

DIAGNOSIS
Decreased serum magnesium

ECG:
• Prolonged QT interval, Prolonged PR, QRS widening
  Increases risk of developing V tach, Torsades de pointes
HYPO MAGNESEMIA

MANAGEMENT

• *Oral Magnesium:*

• *IV Magnesium Sulfate: drug of choice for torsades de pointe* or severe
HYPERMAGNESEMIA

RARE. MC cause is renal insufficiency or overcorrection of hypomagnesemia

• Acute renal failure

• Ingestion of Magnesium substances (ex. Vitamins, antacids)

• Excess IV magnesium administration (ex asthma, eclampsia, Torsades de pointes, arrhythmias)

• Adrenal sufficiency, milk alkali syndrome, Lithium
HYPERMAGNESEMIA

CLINICAL MANIFESTATIONS

- **Neurological**: nausea, vomiting, skin flushing, muscle weakness, lightheadedness, AMS

- **Cardiac**: arrhythmias

MANAGEMENT

- IV fluids + Furosemide (Lasix)

- Calcium Gluconate* 
  antagonizes toxic effects of Mg
HYPOCALCEMIA

ETIOLOGIES:
• Hypoparathyroidism (MC)*, hepatorenal disease, vitamin D deficiency, albumin deficiency.

CLINICAL MANIFESTATIONS:
• Tetany (Chvostek, Trousseau), paresthesias, diarrhea, CHF, syncope, seizures, dry skin, psoriasis

DIAGNOSIS:
• ↓Calcium, ±↑Phos. ECG: prolonged QT*

MANAGEMENT:
• Calcium + Vit D
• IV Ca gluconate: if severe
HYPERCALCEMIA

CLINICAL MANIFESTATIONS

• **Stones:** *kidney stones* (hypercalcuria ⇒ Calcium oxalate & phosphate stones), polyuria, nocturia, nephrogenic diabetes insipidus

• **Bones:** *painful bones, fractures* (bone remodeling).

• **Abdominal groans:** *ileus, constipation*, nausea, vomiting

• **Psychic moans:** weakness, fatigue AMS, ↓*DTR*, depression or psychosis may develop c elevated calcium levels. Blurred vision.
HYPERCALCEMIA

- 90% Hyperparathyroidism or malignancy!!

- **PTH**: 1<sup>ry</sup> hyperparathyroidism, Men I, IIA
- **PTH ind**: malignancy, granulomatous, meds

**DIAGNOSIS:**
- intact PTH, Calcium, (PTH-related protein, vitamin D)

**MANAGEMENT:**
- *IV fluids + loop diuretics, bisphosphonates*, calcitonin
- Steroids: if granulomatous, ↑vit D
<table>
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<th>Anion Gap Metabolic Acidosis</th>
<th>Non-Gap Metabolic Acidosis</th>
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</thead>
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<tr>
<td><strong>“MUDPIILERS”</strong></td>
<td><strong>“HARDUPS”</strong></td>
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<tr>
<td>Methanol</td>
<td>Hyperalimentation</td>
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<td>Uremia</td>
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<td>DKA/Alcoholic KA</td>
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<td>Propylene glycol</td>
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<td>Isoniazid, Infection</td>
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<td>Lactic Acidosis</td>
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<td>Rhabdo/Renal Failure</td>
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<td>Salicylates</td>
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*Too much acid or little Bicarbonate*

*Too much acid or little Bicarbonate*
<table>
<thead>
<tr>
<th>Acute Respiratory Acidosis</th>
<th>Metabolic Alkalosis</th>
<th>Respiratory Alkalosis</th>
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</thead>
<tbody>
<tr>
<td><strong>anything that causes hypoventilation, i.e.</strong></td>
<td><strong>“CLEVER PD”</strong></td>
<td><strong>“CHAMPS”</strong></td>
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<tr>
<td>“CHAMPP”</td>
<td><strong>Contraction</strong></td>
<td><strong>anything that causes hyperventilation, i.e.:</strong></td>
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<td><strong>CNS depression</strong></td>
<td><strong>Licorice</strong></td>
<td><strong>CNS disease</strong></td>
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<td>– (drugs/CVA)</td>
<td><strong>Endo</strong> (Ex Conn’s, Cushing’s)</td>
<td><strong>Hypoxia</strong></td>
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<td><strong>Hemo/Pneumothorax</strong></td>
<td><strong>Vomiting</strong></td>
<td><strong>Anxiety</strong></td>
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<td><strong>Airway Obstruction</strong></td>
<td><strong>Excess Alkali</strong></td>
<td><strong>Mech Ventilators</strong></td>
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<td><strong>Myopathy</strong></td>
<td><strong>Refeeding Alkalosis</strong></td>
<td><strong>Progesterone</strong></td>
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<td><strong>Pneumonia</strong></td>
<td><strong>Post-hypercapnia</strong></td>
<td><strong>Salicylates/Sepsis</strong></td>
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<td><strong>Pulmonary Edema</strong></td>
<td><strong>Diuretics</strong></td>
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<tr>
<td><strong>Anything that decreases respiration</strong></td>
<td><strong>Little acid or too much bicarbonate</strong></td>
<td><strong>Anything that causes hyperventilation</strong></td>
</tr>
</tbody>
</table>
ABG

STEP 1:
• LOOK AT PH

STEP 2:
• LOOK AT PCO2

STEP 3:
• LOOK AT ANION GAP
ABG’S

ACID BASE MNEMONIC (ROME)

R - Respiratory

RO - Opposite

pH \uparrow \quad \text{PCO}_2 \downarrow \quad \text{Alkalosis}

pH \downarrow \quad \text{PCO}_2 \uparrow \quad \text{Acidosis}

O - Opposite

ME - Metabolic

M - Equal

pH \uparrow \quad \text{HCO}_3 \uparrow \quad \text{Alkalosis}

pH \downarrow \quad \text{HCO}_3 \downarrow \quad \text{Acidosis}