Renal Board Review

Brenda Shinar, MD
Question 1.

- Answer: A; Add chlorthalidone
Over 115/75 mm Hg, for every increase in systolic BP by 20 mm Hg or every increase in diastolic blood pressure by 10 mm Hg, the risk of cardiovascular death doubles!
Treatment of Hypertension

• Make the diagnosis
  – Measure correctly
  – Ambulatory monitoring/Home monitoring
  – End organ damage/CV risks

• Look for modifiable risk factors
  – Obesity, sedentary lifestyle, alcohol use, drugs, dietary factors

• Think about secondary causes
  – RAS, OSA, endocrine, coarctation, renal disease

• Pick your medication(s)
  – First line for AA, Caucasian, underlying conditions

• Treat to goal
  – Add synergistic agents, avoid side effects

Resistant hypertension:
Blood pressure that is uncontrolled on 3 medications from different classes, at optimal dose, *one of which is a diuretic* OR controlled on 4 or more medications

• On lisinopril, nifedipine, atenolol
• ADD thiazide (GFR > 30) or loop diuretic (GFR <30)
Question 2.

- Answer: D; Lisinopril
Treating Stage 1 hypertension in African Americans

- Thiazide diuretics and calcium channel blockers (amlodipine and diltiazem) are first line treatment for blood pressure management even in the setting of diabetes for black patients!

- Black patients with CKD can start with ACEI as first line!

Beta blockers are never first line antihypertensive drugs!
Question 3.

- **Answer: D; Continue clinical observation**
Manage HTN in a patient who is over age 60.

**Classification**

<table>
<thead>
<tr>
<th>Blood Pressure Classification</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and &lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120–139</td>
<td>or 80–89</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140–159</td>
<td>or 90–99</td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>≥160</td>
<td>or ≥100</td>
</tr>
</tbody>
</table>

**Goals of Therapy**

**Recommendations from recent guidelines**

<table>
<thead>
<tr>
<th></th>
<th>JNC 8 panel</th>
<th>ASH/ISH</th>
<th>JNC 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>&lt;140/90</td>
<td>&lt;140/90</td>
<td>&lt;140/90</td>
</tr>
<tr>
<td>Elderly</td>
<td>&lt;150/90 (≥60 y)</td>
<td>&lt;150/90 (≥80 y)</td>
<td>&lt;130/80</td>
</tr>
<tr>
<td>DM</td>
<td>&lt;140/90</td>
<td>&lt;140/90</td>
<td>&lt;130/80</td>
</tr>
<tr>
<td>CKD</td>
<td>&lt;140/90</td>
<td>&lt;140/90</td>
<td>&lt;130/80</td>
</tr>
</tbody>
</table>
Question 4.

• Answer: B: Plasma aldosterone-plasma renin activity ratio
Understand the differential diagnosis of resistant hypertension

**Definition of resistant hypertension:**
The failure to reach blood pressure goal despite the **concurrent use of 3 antihypertensive agents of different classes** at maximal tolerated doses, with one of the agents being a **diuretic.** (JNC 7)

OR

Patients whose blood pressure is controlled on 4 or more agents (AHA)

---

**Approach to Resistant HTN**

- Establish diagnosis
- Is there white coat hypertension?
- Is the patient adherent to medical program?
- Is the patient taking interfering substances?
- Is there an identifiable (secondary) cause?
- Optimize and intensify pharmacologic therapy
- Refer to hypertension specialist
- Experimental therapies? (currently none FDA approved...)

**Indications for Further Evaluation for Secondary Causes**

- Abnormal labs - hypercalcemia, hypokalemia with kaliuresis...
- Abrupt onset
- Age < 30 or > 55 years
- Resistant hypertension
- Systolic-diastolic bruits in epigastrium or lateralizing over a kidney
Primary Hyperaldosteronism:
60%+ of patients have NORMAL K level

• ALDOSTERONE HIGH
• RENIN LOW
• AR RATIO >25 is suggestive BUT NOT diagnostic
• Metabolic alkalosis and hypokalemia MAY OR MAY NOT be present

• 8 AM draw
• OFF spironolactone or eplerenone for 6 weeks
• Possibly off ACEI

• NO CONFIRMATION test needed:
  – Spontaneous hypokalemia
  – Undetectable renin level
  – Aldosterone >30 ng/dL
Question 5.

• Answer: D; Sevelamer
CURRENT CHRONIC KIDNEY DISEASE (CKD) NOMENCLATURE USED BY KDIGO

CKD is defined as abnormalities of kidney structure or function, present for > 3 months, with implications for health. CKD is classified based on Cause, GFR category (G1-G5), and Albuminuria category (A1-A3), abbreviated as CGA.

Prognosis of CKD by GFR and albuminuria category

<table>
<thead>
<tr>
<th>Persistent albuminuria categories</th>
<th>Description and range</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>Normal to mildly increased</td>
</tr>
<tr>
<td>A2</td>
<td>Moderately increased</td>
</tr>
<tr>
<td>A3</td>
<td>Severely increased</td>
</tr>
<tr>
<td>≥30 mg/g &lt;3 mg/mmol</td>
<td>30-300 mg/g 3-30 mg/mmol</td>
</tr>
<tr>
<td>&gt;300 mg/g &gt;30 mg/mmol</td>
<td></td>
</tr>
</tbody>
</table>

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012

<table>
<thead>
<tr>
<th>GFR categories (mL/min/1.73 m²)</th>
<th>Description and range</th>
</tr>
</thead>
<tbody>
<tr>
<td>G1</td>
<td>Normal or high ≥90</td>
</tr>
<tr>
<td>G2</td>
<td>Mildly decreased 60-89</td>
</tr>
<tr>
<td>G3a</td>
<td>Mildly to moderately decreased 45-59</td>
</tr>
<tr>
<td>G3b</td>
<td>Moderately to severely decreased 30-44</td>
</tr>
<tr>
<td>G4</td>
<td>Severely decreased 15-29</td>
</tr>
<tr>
<td>G5</td>
<td>Kidney failure &lt;15</td>
</tr>
</tbody>
</table>

Green: low risk (if no other markers of kidney disease, no CKD); Yellow: moderately increased risk; Orange: high risk; Red, very high risk.
US Prevalence of CKD Stages

Pathway of Chronic Kidney Disease - Mineral and Bone Disorder (CKD-MBD)

- **Renal function**
  - Phosphate retention
    - FGF23
    - ↓1,25 D production
    - ↓VDR expression
    - ↑PTH
    - ↓Ca²⁺
    - ↑PO₄
    - Altered parathyroid gland function
      - Hyperplasia
      - Secondary hyperparathyroidism
    - Hyperphosphatemia

**CONSEQUENCES**
- Renal osteodystrophy
- Fractures
- Calcification
- Cardiovascular disease

**Morbidity and mortality**

VDR= vitamin D receptor
PTH= parathyroid hormone
Ca²⁺= calcium ions
PO₄= phosphate
MRB 001 2009
Treat hyperphosphatemia in a patient with CKD

KDIGO guidelines 2016

1. Phosphorus-restricted diet
2. Phosphorus binders
   - Calcium-containing P04 binders:
     • Calcium carbonate (Tums)
     • Calcium acetate (PhosLo)
     • Appropriate for all hypo-calcemic patients and normocalcemic patients without vascular calcification or adynamic bone disease
   - Noncalcium-containing P04 binders:
     • Sevelamer (Renagel, Renvela)
     • Lanthanum (FosRenal)
     • Appropriate for hypercalcemic patients and patients with vascular calcification, adynamic bone disease, or patients on 1,25-OH vitamin D supplements
Management of Anemia in CKD

1) Rule out other causes of Anemia – bleeding, nutritional deficiencies.

2) Once CKD cause of Anemia is established – Evaluate for iron deficiency – check Iron SAT and Ferritin, consier supplemental Fe (oral or IV) as needed, and Evaluate Hgb response

3) If anemia persists consider ESA’s – Do Not Over-treat!!! Hgb >10 is current goal.
Renal Function vs Death, Cardiovascular Events, or Hospitalization Endpoints

Rate of Death From Any Cause (per 100 person-years)*

<table>
<thead>
<tr>
<th>Estimated GFR†</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥60</td>
<td>2</td>
</tr>
<tr>
<td>45-59</td>
<td>4</td>
</tr>
<tr>
<td>30-44</td>
<td>8</td>
</tr>
<tr>
<td>15-29</td>
<td>14</td>
</tr>
<tr>
<td>&lt;15</td>
<td>16</td>
</tr>
</tbody>
</table>

Cardiovascular Events Rate (per 100 person-years)*

<table>
<thead>
<tr>
<th>Estimated GFR†</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥60</td>
<td>0.5</td>
</tr>
<tr>
<td>45-59</td>
<td>2</td>
</tr>
<tr>
<td>30-44</td>
<td>5</td>
</tr>
<tr>
<td>15-29</td>
<td>10</td>
</tr>
<tr>
<td>&lt;15</td>
<td>20</td>
</tr>
</tbody>
</table>

N= 1,120,295

*Age-standardized rates
Question 6.

• Answer: C: Measure urine chloride level
Evaluate a Patient with Hypokalemic Metabolic Alkalosis

**URINE CHLORIDE < 15 (LOW) =**
Chloride Responsive (90%)

- Low effective circulating volume
- Volume depleted (orthostatic, hypotensive)
- **NORMAL** increase in renin, angiotensin, aldosterone
- Urine chloride LOW <15
- Cannot replace K until volume is replaced (GIVE sodium CHLORIDE)

**URINE CHLORIDE >15 (HIGH) =**
Chloride Unresponsive (10%)

- Hypertensive
- Hypervolemic
- **ABNORMAL** increase in aldosterone (Primary hyperaldo) or renin (Secondary hyperaldo)
- Urine chloride HIGH >15
Diagnosis of Metabolic Alkalosis

Once it has been determined that a patient has metabolic alkalosis, the etiology is usually obvious from the history. If there is no pertinent history, then one can assume that the alkalosis is due to one of the three most common causes: 1) vomiting, 2) diuretics, 3) mineralocorticoid excess. To differentiate between these conditions, it is usually helpful to measure the **urinary chloride concentration**.

In causes of metabolic alkalosis associated with a reduction in the ECV, there will be a stimulus for avid Na and Cl reabsorption to replenish extracellular volume. In these setting **urinary Cl should be expected to be very low, less than 25 meq/L**.

Urinary Na is not a reliable measure of extracellular volume in this setting because if the alkalosis is such that not all of the filtered HCO3-can be reabsorbed, then some will be excreted with Na and the urinary Na may be high. Thus, it may appear that the volume status is euolemic or hypervolemic when it is not.

If the urinary Cl is low, indicating a hypovolemic state, then administration of NaCl and water to replenish the extracellular volume should stop the stimulus for aldosterone production and in turn should lead to appropriate excretion of excess HCO3- and improvement of hypokalemia. Thus, leading to correction of the metabolic alkalosis. Such causes of metabolic alkalosis are said to be **saline responsive**. See table below.

In contrast, states of **mineralocorticoid excess are associated with an expanded volume** and sometimes hypertension. The **urinary Cl will be high (> 40 meq/L)**. In these patients, administration of saline would further expand the extracellular volume and worsen hypertension. It would not correct the alkalosis which is primarily due to hypokalemia. Such causes of metabolic alkalosis are said to be **saline resistant**.

<table>
<thead>
<tr>
<th>Urine Cl &lt; 25 meq/L</th>
<th>Urine Cl &gt; 40 meq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Saline Responsive</strong></td>
<td><strong>Saline Unresponsive</strong></td>
</tr>
<tr>
<td>Vomiting or nasogastric suction</td>
<td>Primary mineralocorticoid excess</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Exogenous Alkali load</td>
</tr>
<tr>
<td>Posthypercapnia</td>
<td>Barter's or Gitelman's syndrome</td>
</tr>
<tr>
<td>Cystic Fibrosis</td>
<td>Severe Hypokalemia (K&lt; 2.0)</td>
</tr>
<tr>
<td>Low chloride intake</td>
<td></td>
</tr>
</tbody>
</table>

Causes of saline resistant metabolic alkalosis can further be distinguished based on whether or not the patient is hypertensive. Mineralocorticoid excess states tend to be associated with hypertension while exogenous alkali load, Barter's and Gitelman's syndrome are associated with normal blood pressure.
Question 7.

• Answer: E, Estimated GFR of < 15mL/min
Know how to appropriately dosing medications in acute kidney injury

**Estimation of Creatinine Clearance**
1. Crockcoft-gault
2. MDRD
3. CKD-EPI

All equations require steady-state, stable serum creatinine to be valid.

**Epidemiology**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>N</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATN</td>
<td>337</td>
<td>45</td>
</tr>
<tr>
<td>Prerenal</td>
<td>158</td>
<td>21</td>
</tr>
<tr>
<td>Obstructive</td>
<td>75</td>
<td>10</td>
</tr>
<tr>
<td>Glomerulonephritis</td>
<td>23</td>
<td>3</td>
</tr>
<tr>
<td>AIN</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>Vasculitis</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>Vascular</td>
<td>8</td>
<td>1</td>
</tr>
</tbody>
</table>

**Serum Creatinine in Severe AKI**
Question 8.

- Answer: B: Hypokalemic distal (type 1) renal tubular acidosis
Diagnose hypokalemic distal (type 1) renal tubular acidosis

<table>
<thead>
<tr>
<th></th>
<th>Type 1 RTA (distal)</th>
<th>Type 2 RTA (proximal)</th>
<th>Type 4 RTA (distal)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chloride</strong></td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Bicarbonate</strong></td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td>↓</td>
<td>NL</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Urine pH</strong></td>
<td>High (&gt; 5.5)</td>
<td>Low (&lt;5.5) except with bicarb load</td>
<td>Low (&lt;5.5)</td>
</tr>
<tr>
<td><strong>Etiologies</strong></td>
<td>Chronic hepatitis Amphotericin B Toluene Lithium Sjogren’s; SLE</td>
<td>Multiple Myeloma Metal poisoning Acetazolamide</td>
<td>Diabetes mellitus Sickle cell Spironolactone</td>
</tr>
<tr>
<td><strong>Associations</strong></td>
<td>Nephrolithiasis due to hypercalcuria</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Question 9.

- Answer: B: Chlorthalidone
Manage hypercalciuria in a patient with nephrolithiasis

• Hypercalciuria:
  – >250 mg/24 hours WOMEN
  – >300 mg/24 hours MEN
  – > 200 mg/liter

• WORSENED by:
  – High sodium diet
  – High animal protein diet
  – Loop diuretics

• DO NOT advise a calcium restricted diet, as this increases GI absorption of oxalate, increasing oxaluria

• DO ADVISE:
  – Thiazide diuretic
  – Fluids > 2 liters/day
  – Low purine diet
  – Low sodium diet
Question 10.

• Answer: C; Oral sodium bicarbonate
Treating metabolic acidosis in CKD

Problem List:
- 58 year-old woman
- HTN, CKD stage 3b d/t analgesics
- Amlodipine
- Low serum bicarb, pH 7.36, AG of 12
- Mild asymptomatic NAGMA

Non-AG metabolic acidosis:
- Renal (RTAs) – impaired acid excretion
- GI (diarrhea) – loss of bicarb
- Medications: Lithium, TPN

Our patients history suggests Type IV RTA
1. H/o analgesic nephropathy
2. Normal AG MA
3. Hyperkalemia

Treatment goal:
- Serum bicarb goal 23-29 which reduces risk of CKD progression
- Bicarbonate administration will help correct the acidosis.
- Dose: 0.5 to 1mEq/kg/day
Question 11.

• Answer: A; AL amyloidosis
Diagnose Multiple Myeloma as a cause of acute kidney injury

**Clinical Features of MM:**
- Anemia (NCNC) (80%)
- Bone pain (70%)
- Recurrent infections
  - 25% presenting
  - 75% during disease
- Renal complications (50%)
- Hypercalcemia (25%)
- Renal failure (25%)

**Renal Complications:**
- Tubular Damage
  - Adult Fanconi’s syndrome
  - RTA Proximal Type 2
- Cast Nephropathy *
  - Proteinaceous casts clog the tubules resulting in tubule atrophy
- Glomerulopathy
  - Light chain disease deposition
  - Resulting in *albuminuria*
- Exquisite sensitivity to IV contrast!

*most common
### Amyloidosis: In an apple-green nutshell

<table>
<thead>
<tr>
<th><strong>Primary AL Amyloidosis</strong></th>
<th><strong>Secondary AA Amyloidosis</strong></th>
<th><strong>Hereditary ATTR Amyloidosis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Monoclonal protein related to plasma cell dyscrasia</td>
<td>• <em>Polyclonal protein</em> related to inflammatory state; rheumatologic or infectious</td>
<td>• <em>Mutation in the transthyretin gene</em> making abnormal proteins that infiltrate organs</td>
</tr>
<tr>
<td>• SPEP/IFE, UPEP/IFE, free light chains</td>
<td>• Juvenile RA, anklyosing spondylitis, psoriatic arthritis, IBD</td>
<td>• Cardiac and neurologic involvement more common than renal involvement</td>
</tr>
<tr>
<td>• Bone marrow positive for plasma cell proliferation</td>
<td>• Familial Mediterranean Fever (60% of cases in Turkey)</td>
<td>• NEGATIVE SPEP/IFE etc for monoclonal proteins</td>
</tr>
<tr>
<td>• Monoclonal proteins infiltrate tissues; heart, kidney, GI tract, pleura, nerves (peripheral and autonomic)</td>
<td>• NEGATIVE SPEP/IFE etc for monoclonal proteins</td>
<td>• Rare to have kidney involvement</td>
</tr>
<tr>
<td>• Kidney involvement may result in nephrotic proteinuria (albumin)</td>
<td>• Kidney disease may result in nephrotic proteinuria (albumin)</td>
<td></td>
</tr>
</tbody>
</table>
Question 12.

• Answer: A: Interstitial nephritis
Diagnose Acute Interstitial Nephritis

Clinical Presentation:
- Fever, rash, eosinophilia, and elevated creatinine (10%)
- UA: WBC, WBC casts
- NEGATIVE CULTURE (sterile pyuria)
- Eosinophiluria (sensitive but not specific)
- < 2 gm/24 hr proteinuria

Causes: Drugs, Infections, Illnesses
- Antibiotics (B-lactam, cephalosporins, quinolones, Bactrim, rifampin)
- NSAIDS * (nephrotic proteinuria) with minimal change disease on biopsy
- Thiazides
- Proton Pump Inhibitors
- Phenytoin
- Allopurinol
- 5-aminosalicylates
Question 13.

• Answer: D; Continued current therapy
Diagnose and manage infection-related glomerulonephritis

- 24 y/o man IVDU
- H/o previous staph endocarditis with prolonged antibiotics
- Recent dx of MRSA endocarditis on IV vanco
- Worsening renal function

**Laboratory Studies**
- Creatinine 2.8
- Active UA with protein, RBCs and RBC casts
- Low C3, nL C4
- Negative cryoglobulins
- Renal U/S with Doppler: normal kidneys

**DDx:**
- Acute interstitial nephritis
- Drug induced nephrotoxicity
- Infection related GN
- Septic emboli

Clinical picture and labs suggest infection related GN.

Treatment is to continue treating the infection.
Manage Post-Infectious Glomerulonephritis (PIGN)

- MANY bacteria, viruses and parasites can cause PIGN
- Most common nephritogenic strains of strep and staph
- Rapid onset of hypertension, oliguria, erythrocyte casts, and edema, **LOW C3**

**Pathophysiology:**
- Immune complex-mediated disease
- Complexes deposit in glomerulous and active complement, recruiting inflammatory cells and causing proliferative GN
- Common in non-strep infections (antigen is the infection)

Why isn’t Vancomycin the culprit?
Drug induced tubular toxicity occurs 7 -10 days after Abx initiation and there is usually no cells in urine sediment.
Question 14.

• Answer: B; Ethylene glycol poisoning
Diagnose ethylene glycol poisoning

**AGMA**

**Methanol**
Sources: windshield wiper fluid, moonshine, perfumes
Effects: formic acid causes vision changes (blindness), “snowfield vision” (toxic to mitochondrial of retina and optic nerve), basal ganglia effects and pancreatitis
Labs: Anion gap + Osmolar gap

**Toxic alcohol**

**Isopropyl Alcohol**
Sources: rubbing alcohol/hand sanitizer
Effects: hemorrhagic gastritis, fruity breath
Labs: Osmolar gap ONLY (no anion gap), ketones

**NAGMA**

**Ethylene Glycol**
Sources: antifreeze
Effects: oxalic acid causes cardiac and neuro effects
Labs: Anion gap + Osmolar gap, calcium oxalate crystals deposit in renal tubules, elevated creatinine, false lactic acid elevation from blood gas analyzer (serum lactate is normal)
Ethylene Glycol Poisoning

**Work up:**
- Check blood gas and electrolytes
- If +AGMA, check ketones, lactic acid and calculate osmolar gap

Osmolar gap = measured – calculated osmolarity

Calculated osmolarity = (Na x 2) + (BUN/2.8) + (Glucose/18) = 287,
Measured = 314 - 287 = 27 gap.

>10 suggest the presence of an osmotically active substance
>20 is usually due to toxic alcohol ingestion, >50 for sure!

**Treatment:**
- Fomepizole (blocks alcohol dehydrogenase and autometabolizes)
- Ethanol administration is complicated by inebriation, erratic metabolism and hypoglycemia
- HD removes alcohol and toxic metabolites
- Dialyze if high levels of toxic alcohols, severe AGMA, electrolyte disturbances
Question 15.

• **Answer: A: Cryoglobulinemia associated with Hepatitis C**
Diagnose Hepatitis C virus-associated membranoproliferative glomerulonephritis due to cryoglobulins

- Occurs in up to 20% of patients with chronic Hepatitis C
- Presents as membranoproliferative glomerulonephritis or mixed cryoglobulinemia (skin, kidney, and nerve involvement)
- 1/3 relapsing dz with progression to ESRD
- Low complement (C4)
- + Rheumatoid factor
- TREAT Hep C virus

EXTRA-HEPATIC MANIFESTATIONS OF HEPATITIS C INFECTION:

1. Membranoproliferative GNritis
2. Mixed Essential Cryoglobulinemia
3. Lichen Planus
4. Porphyria Cutanea Tarda
Question 16.

- Answer: C; Hemoglobinuria
Evaluation of Red Urine

Hemoglobinuria due to Intravascular hemolysis:

- Microangiopathic hemolytic anemia
- Transfusion reactions
- Mechanical shear from valves
- Infections
  - (Clostridial sepsis, Malaria)
- Paroxysmal Nocturnal Hemoglobinuria (PNH)
- Hypotonic fluid infusions
- Exposure to compounds with high oxidant potential (copper poisoning, Wilson’s disease)
Question 17.

- Answer: B; HIV antibody test
Diagnose Focal Segmental Glomerulosclerosis: the most common renal cause of nephrotic syndrome in US (36-80%)

**Primary Disease:**
Podocyte damage similar to minimal change dz

**Secondary Disease Associations:**
- Morbid obesity
- Heroin use
- HIV
- Vesicoureteral reflux

**Risk factors for progression to ESRD:**
- Black race
- >2 gm/24 hr proteinuria
- Low GFR
- BMI>27
- Hypertension

**Clinical Manifestations:**
- Acute onset of nephrotic syndrome with hematuria, renal failure and hypertension in primary disease;
- Asymptomatic subnephrotic to nephrotic proteinuria in secondary disease

**Management:**
- Immunosuppression with steroids or calcineurin inhibitors in primary disease
- ACEI +/- ARB in primary and secondary disease
- BP goal < 125/75 mm Hg
Question 18.

- Answer: B: Membranous nephropathy
Diagnose Membranous Nephropathy:
the second most common renal cause of nephrotic syndrome

**Primary disease:**
- Immune complexes of IgG react with antigens in the outer aspect of GBM
- Antibody to PLA2

**Secondary disease associations:**
- Malignancy
- Hep B and C
- NSAIDS

**Clinical manifestations:**
- Nephrotic syndrome with preserved GFR
- Renal vein thrombosis
- Hypercoagulability

**Risk factors for progression to ESRD:**
- Male gender
- Age>50
- Persistent proteinuria > 4g/24 h over 6 months
- Declining GFR

**Management:**
- ACE and/or ARB
- BP goal < 125/75 mm Hg
- Persistent proteinuria > 4g/24 hr
- Cyclophosphamide, corticosteroids, or calcineurin inhibitor with possible need for rituxan
Question 19.

• Answer: B; Hyperglycemia
Patient has hypertonic hyponatremia!
Correction factor: 1.6mEq/L decrease in the serum sodium level per every 100mg/dL of plasma glucose over 100mg/dL.
Treatment: fluids and treat hyperglycemia.
Algorithm for hypotonic hyponatremia

**Diagnostic Algorithm for Hyponatremia**

**Assessment of ECF volume status**

**Hypovolemia**
- Total body water
- Total body Na⁺
  - Urine Na⁺ < 20 mmol/L
  - Urine osmolality > 100 mmol/l
- Non-renal loss
  - Vomiting
  - Diarrhea
  - Sweating
  - Third-spacing
  - Burns
- Renal loss
  - Diuretics
  - Osmotic diuresis
  - Salt-losing nephritis
  - Cerebral salt-wasting
  - Addison disease

**Euvolemia**
- Total body water
- Total body Na⁺
  - Urine Na⁺ > 20 mmol/L
  - Urine osmolality > 100 mmol/l
  - Urine osmolality > 100 mmol/kg
- SIADH
  - Drug-induced
  - Tumors
  - CNS disorders
  - Lung diseases
  - Hypothyroidism
  - Glucocorticoid deficiency
- Primary polydipsia
  - Low solute intake
- Post surgery
- Reset osmostat

**Hypervolemia**
- Total body water
- Total body Na⁺
  - Urine Na⁺ < 20 mmol/L
  - Urine osmolality > 100 mmol/l
- Acute or chronic renal failure
- Nephrosis
- Cirrhosis
- Heart failure
Manage asymptomatic hypotonic hyponatremia

**Fig. 3 - Treatment algorithm for hyponatremia: severe hypotonic hyponatremia (serum sodium <125 mmol/L). AVP = arginine vasopressin; ECF = extracellular fluid.**
Question 20.

- Answer: B; Substitute labetalol for lisinopril
Manage HTN in a woman of childbearing age

Normal physiology in pregnancy
- Blood pressure lowers
- Plasma volume increases
- GFR increases
- Renal pelvis, calices, and ureters dilate
- Increased risk for pyelo
- Hyperventilation causes resp alkalosis with increased renal bicarb excretion

Four Major Hypertensive Disorders in Pregnancy
- Chronic HTN:
  Pre-existing HTN or HTN before 20th week of gestation
- Gestational HTN:
  HTN after 20th week with no proteinuria and no prior h/o HTN (resolves by 12 weeks post-partum)
- Preeclampsia:
  HTN + proteinuria after 20th week of gestation or signs of end-organ dysfunction (platelet count <100,000/microliter, serum Cr >1.1 mg/dL or doubling, elevated transaminases to 2x normal)
- Preeclampsia complicating chronic HTN:
  Worsening HTN + proteinuria after 20th week of gestation with history of controlled, chronic HTN

Pearls in Pregnancy “20 weeks”:

ACEI, ARB, and Renin-Inhibitors are CONTRAINDICATED in pregnancy!!