CRUNCH TIME

ENDOCRINE

Acute Adrenal Crisis

Vanessa Cardy, MD; Gita Pensa, MD; and Mel Herbert, MD

Background

- Shock that is not responsive to fluid
- 3 classic presentations:
    - Infants
    - Patients with recent steroid withdrawal
    - Patient with primary Addison's disease with physiologic stress

Clinical Findings

- Shock
- Cardiovascular collapse
- Hypotension
- Hyponatremia
- Hyperkalemia
- Hypoglycemia
- Low cortisol levels
- Abnormal adrenocorticotropic hormone (ACTH) stimulation test

Management

- IV fluids: D5NS (5% dextrose in normal saline) is fluid of choice in cases with hyponatremia and hypoglycemia
- Hydrocortisone
- Dexamethasone 4-6 mg IV if potassium <6 mmol/L
- Address hyperkalemia
- If still hypotensive, consider vasopressors

How You Will Be Tested

- Make sure you remember electrolyte association (hyponatremia, hyperkalemia)
- Shock refractory to fluids
- Remember the 3 classic presentations

Adrenal Insufficiency

Jessica Mason, MD, and Mel Herbert, MD

Background

- Adrenal glands have difficulty producing cortisol and/or aldosterone
- Primary: adrenal malfunction
    - Autoimmune: Addison's disease (most common cause)
    - Infection/infiltration (tuberculosis most common)
    - Hemorrhage
        - Hemorrhage into adrenals (Waterhouse-Friderichsen syndrome)
- Secondary: pituitary issue
    - Hemorrhage into pituitary (Sheehan syndrome after delivery of infant)
    - Most common cause: steroid withdrawal
    - Iatrogenic adrenal suppression from chronic steroid use, which leads to native gland suppression/malfunction

Clinical Findings

- Shock
- Generalized malaise
- Fatigue
- Abdominal pain
- Skin hyperpigmentation (Addison's disease)
- Cushingoid appearance (if chronically on steroids)
- Hyperkalemia
- Hyponatremia
- Elevated blood urea nitrogen
- Hypoglycemia
- Random cortisol level <15 µg/dL with symptoms is diagnostic

Management

- D5NS (5% dextrose in normal saline)
- Hydrocortisone 100 mg IV push and then 100 mg q6-8h
- Chronic: prednisone or hydrocortisone by mouth

How You Will Be Tested

- Shock that is not responsive to fluids
- Most common cause is autoimmune
- Hyperpigmentation in Addison's disease
- Memorize metabolic derangements and treatment with hydrocortisone

Adrenal Tumors & Pheochromocytoma

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- Pheochromocytoma
    - Chromaffin cell tumor of the adrenals that secrete catecholamine
    - Can also secrete glucocorticoids causing Cushing syndrome
    - Rule of 10s:
        - 10% extra-adrenal
        - 10% bilateral
        - 10% malignant
        - 10% familial
        - 10% occur in children
        - 10% have multiple tumors

Clinical Findings

- Pheochromocytoma
    - Episodic headache, sweating, and tachycardia
    - Weight loss
    - Severe hypertension, stroke, myocardial infarction
    - Gold standard for diagnosis: 24-h urine collection looking for catecholamine secretion
    - Elevated plasma metanephrines

Management

- Pheochromocytoma
    - Assess for end-organ damage
    - Alpha blockade to control hypertension with phentolamine
    - Alpha blockade before any beta blockade
    - Surgical consultation

How You Will Be Tested

- Given a case of an individual with episodic headache, tachycardia, and other symptoms of catecholamine surge
- Alpha blockade before beta blockade (risk of unopposed alpha causing increase in blood pressure) when treating

Approach to Respiratory Acidosis

Jessica Mason, MD, and Mel Herbert, MD

Background

- Acidosis = pH <7.35

Clinical Findings

- Hypoventilation
    - (Decreased respiratory rate)
    - Obstructive disease (chronic obstructive pulmonary disease, asthma)
    - Pickwickian syndrome (obesity hypoventilation)
    - Neuromuscular disorder

Management

- Help patient ventilate and get rid of the excess CO_2

How You Will Be Tested

- You will be given a blood gas and asked to identify a respiratory acidosis (partial pressure of CO_2 [PCO_2] >45)

Approach to Metabolic Acidosis

Jessica Mason, MD; Mel Herbert, MD; and Jessie Werner, MD

Background

- Low pH (<7.35), bicarbonate <20
- MUDPILESCAT
    - **M**ethanol
    - **U**remia
    - **D**iabetic ketoacidosis
    - **P**henformin or **P**aracetamol
    - **I**soniazid or **I**ron
    - **L**actic acidosis
    - **E**thylene glycol
    - **S**alicylates
    - **C**arbon monoxide or cyanide poisoning
    - **A**lcoholic ketoacidosis
    - **T**oluene (also produces hypokalemia)
- Ethanol and isopropyl alcohol cause an osmolar gap but not an anion gap
- Elevated osmolar gap from all toxic alcohols, uremia, ketoacidosis, acetone, and mannitol
- Causes of non-anion-gap acidosis
    - Gastrointestinal losses
    - Hyperchloremic acidosis from resuscitation with normal saline
- Bicarbonate on a blood gas is calculated, whereas bicarbonate level from a chemistry panel is measured; use the measured level in all subsequent calculations

Clinical Findings

- Depends on the underlying cause of the acidosis

Management

- Treat the underlying causes

How You Will Be Tested

- Calculating anion gap (Na - Cl - HCO_3): anion gap >10 is elevated
- Calculate osmolar gap (measured osm - calculated osm)
    - Calculated osm (2× Na) + glucose/18 + blood urea nitrogen/2.8 + ethanol/4.6
- Winter's formula:
    - Assess respiratory response to metabolic acidosis
    - Partial pressure of CO_2 (PCO_2) = 1.5 × HCO_3 + 8; if within 2 of this number, response is appropriate

Approach to Alkalosis

Jessica Mason, MD, and Mel Herbert, MD

Background

- Primary metabolic alkalosis
    - Vomiting (most common)
    - Diuretics
- Primary respiratory alkalosis
    - Breathing too fast
    - Anxiety/panic attack
    - Hypoxemia and air hunger
    - Salicylate toxicity

Clinical Findings

- Metabolic alkalosis
    - Vomiting
- Respiratory alkalosis
    - Can have concomitant metabolic acidosis
    - Hyperventilation

Management

- Metabolic alkalosis - fluids
- Treat the underlying cause

How You Will Be Tested

- You will be asked to identify a potential cause of a patient's metabolic alkalosis and to manage it

Parathyroid Disorders

Jessica Mason, MD; Mel Herbert, MD; and Jessie Werner, MD

Background

- Parathyroid hormone regulates calcium and phosphate in the body
    - Regulates calcium via
        - Gastrointestinal absorption
        - Renal excretion
        - Osteoclast activity
    - Regulates phosphate via
        - Renal excretion

Clinical Findings

- Hypoparathyroidism > hypocalcemia
    - Can occur post-thyroidectomy
- Hyperparathyroidism > hypercalcemia
    - Primary: Increased secretion of hormone from the gland
    - Secondary: Seen in renal failure or vitamin D deficiency - hyperphosphatemia and hypocalcemia
    - Tertiary: Hyperphosphatemia, hypercalcemia, and high parathyroid hormone

Management

- Treat the underlying metabolic derangement

How You Will Be Tested

- Electrolyte abnormalities with hypo- and hyperparathyroidism
- Know the presentation and treatments of hyper- and hypocalcemia

Hyper & Hypocalcemia

Jessica Mason, MD, and Mel Herbert, MD

Background

- Hypercalcemia causes
    - Hyperparathyroidism
    - Malignancy
    - Granulomatous disease
    - Thiazide diuretics
    - Lithium
- Hypocalcemia
    - Hypoparathyroidism
    - Vitamin D deficiency
    - Sepsis
    - Renal failure
    - Pancreatitis (causes saponification of pancreas)
    - Rhabdomyolysis (complexes with phosphate)

Clinical Findings

- Hypercalcemia
    - Stones (nephrolithiasis)
    - Groans (abdominal pain)
    - Bones (bone pain from increased osteoclastic resorption)
    - Psychiatric moans (altered mental status)
    - Shortened QT interval and Osborn waves on ECG
- Hypocalcemia
    - Chvostek sign - facial twitching
    - Trousseau sign - carpopedal spasm
    - Paresthesia
    - Hyperreflexia
    - ECG can show long QT interval, which can evolve into torsades de pointes

Management

- Hypercalcemia
    - IV fluids
    - Treat underlying causes
    - Bisphosphonates
    - Steroids
- Hypocalcemia
    - Order a magnesium level as well (can cause resistance to parathyroid hormone over time)
    - Replete with IV calcium (10 mL over 20 min of IV calcium gluconate)
    - If able to tolerate oral (PO) intake, can give PO calcium

How You Will Be Tested

- Pseudohypocalcemia (total calcium low, ionized calcium high); if albumin is low, will have low total calcium
- Symptoms of hyper- and hypocalcemia

Cushing's Syndrome

Vanessa Cardy, MD, and Stuart Swadron, MD

Background

- Hypercortisolism (excess of glucocorticoids)
- Most common cause (exogenous steroid administration)
- Also have tumor or ectopic production of glucocorticoids

Clinical Findings

- Weight gain
- Moon facies
- Hirsutism
- Flushing
- Fatigue
- Menstrual cycle changes
- Emotional lability
- Sexual dysfunction
- Psychosis
- Possible hypertension
- Truncal obesity
- Buffalo hump (deposition of adipose tissue at base of neck)
- Thinning of skin (abdominal striae)
- Osteoporosis
- Metabolic derangements
    - Hypokalemia
    - Hypochloremia
    - Hyperglycemia

Management

- Treat tumors medically or surgically
- Gradually withdraw steroids to prevent addisonian crisis

How You Will Be Tested

- Know how to recognize it
- Know patients are at high risk for infection (effectively immunocompromised)
- Image of patient with classic cushingoid features

Type 1 Diabetes - Treatment

Gita Pensa, MD, and Jessica Mason, MD

Background

- Destruction of pancreatic beta cells (no endogenous insulin production)
- Can present in adults or kids

Clinical Findings

- Duration of illness leads to worse complications
- Vasculopathy
- Infectious diseases
- Neuropathy
- Nephropathy
- Ophthalmologic complications
- Poor wound healing
- Foot wounds

Management

- Glucose control

How You Will Be Tested

- Have a high index of suspicion for severe illness

Type 1 Diabetes - Management

Gita Pensa, MD, and Jessica Mason, MD

Background

- Insulin (short-, medium-, and long-acting)
- Insulin pumps give continuous subcutaneous infusion of fast-acting insulin
- Pump should be removed before MRI

Clinical Findings

- Hypo- or hyperglycemia
- Pump failure
    - Diabetic ketoacidosis
- Infection at pump site or lipodystrophy (scarring) can affect absorption
- If on amylin may be a cause of hypoglycemia

Management

- Administer insulin
- Pramlintide - synthetic form of amylin that can assist with glucose management

How You Will Be Tested

- Know that management of type 1 diabetes is insulin

Type 2 Diabetes - basics

Gita Pensa, MD, and Jessica Mason, MD

Background

- Decreased insulin production
- Increased insulin resistance
- Gradual onset of symptoms

Clinical Findings

- Cardiac disease
- Neuropathy
- Ophthalmic complications
- Nephropathy
- Gastroparesis
- Fatigue
- Polyuria
- Polydipsia
- Weight loss

Management

- Oral medications with or without insulin
- Metformin
    - Often causes gastrointestinal side effects
    - Can cause lactic acidosis (especially in patients with renal disease)
    - Hold for 48 h after contrast administration
    - If creatinine <1.4 and patient stable, consider starting in the ED
- Sulfonylurea
    - Causes significant hypoglycemia
- Liraglutide
    - Can cause pancreatitis
- Check electrolytes and hemoglobin A1c

How You Will Be Tested

- Know the side effects of some common medications
- Know the common long-term complications associated with type 2 diabetes

Diabetic Ketoacidosis (DKA)

Jennifer Farah, MD; Stuart Swadron, MD; and Jessie Werner, MD

Background

- Diabetic ketoacidosis (DKA) results from a lack of insulin or increased resistance to insulin
- Breakdown of fatty acids for energy leading to ketone production
- Most common cause is insulin non-compliance
- Consider secondary insult
    - Urinary tract infection
    - Pneumonia
    - Myocardial infarction
    - Pregnancy
    - Surgery
    - Trauma
- Total body potassium depleted despite serum elevation

Clinical Findings

- Physical exam
    - Polydipsia
    - Polyuria
    - Kussmaul breathing
        - Classic air-hungry breathing with massive tidal volume
    - Anion gap metabolic acidosis
    - Urine ketones (only measures acetoacetate)
    - Serum ketones (also measures beta hydroxybutyrate)

Management

- Fluids
    - If glucose <250 mg/dL, consider dextrose-containing fluids
- Always check potassium before insulin infusion (0.1 units/kg/h)
- If low, hold insulin
- Bicarbonate only used when pH <7.0

How You Will Be Tested

- Know the difference between DKA and hyperosmolar hyperglycemic state (HHS)
- Have a general understanding of the secondary insults that can exacerbate DKA
- Potassium levels
    - 3.3-5.3: Replenish potassium while administering insulin
    - <3.3: Do not begin insulin administration until potassium replenished
    - >5.3: Can begin insulin and fluids normally
- Glucose levels
    - At 200 mg/dL, use dextrose-containing fluids and decrease insulin infusion rate

Hyperosmolar Hyperglycemic State

Jennifer Farah, MD; Stuart Swadron, MD; and Jessie Werner, MD

Background

- Hyperosmolar hyperglycemic state (HHS) has a significantly higher mortality than diabetic ketoacidosis (DKA)
- Patients with HHS are more severely dehydrated than in DKA
- Significant trigger (myocardial infarction, stroke…)

Clinical Findings

- Lethargy
- Dehydration
- Sequelae of underlying stressor
- Labs
    - Glucose >1,000 mg/dL
    - No ketones due to ketoacidosis; can have scant ketones due to dehydration

Management

- Fluids
- Insulin (not needed to prevent production of ketoacids) and started later
- Electrolyte replacement
- Check for underlying stressor

How You Will Be Tested

- Know the differences between hyperglycemic hyperosmolar nonketotic coma (HONK)/HHS and DKA
- On the test, lab work will show absence of serum ketones

Fluid Overload & Volume Depletion

Gita Pensa, MD, and Jessica Mason, MD

Background

- Either too much fluid to take off or not enough and replenish while correcting electrolytes
- Volume depletion
    - Can cause poor perfusion
- Fluid overload
    - Too much volume
    - Iatrogenic (too much IV fluids)
    - Congestive heart failure
    - Nephrotic syndrome
    - Cirrhosis

Clinical Findings

- Volume depletion
    - Signs of poor perfusion
    - Dry mucous membranes
    - Hypotension
    - Several pathologies can cause losses
        - Gastrointestinal losses, skin losses, hemorrhage, renal (diuretics polyuria), third spacing in crush injury or sepsis
- Volume overload
    - Edema
    - Ascites

Management

- Correct electrolyte abnormalities
- Assess fluid status through labs and physical exam
- Volume depletion
    - Replenish fluids
- Volume overload
    - Remove fluids

How You Will Be Tested

- Recognize the physical exam findings of volume overload and depletion
- Know the underlying pathologies of volume overload and depletion

Hypomagnesemia

Vanessa Cardy, MD; Gita Pensa, MD; and Mel Herbert, MD

Background

- 60% of total body magnesium stored in bone, only 1% is extracellular
- Magnesium follows calcium and phosphate
- Can also affect potassium metabolism
- Caused by decreased absorption through the gastrointestinal tract (poor oral intake, pancreatitis, diarrhea)
- Increased renal losses (diuresis, nephrotoxins, congenital renal defects)

Clinical Findings

- Altered mental status
- Seizure
- Lethargy
- Carpopedal spasms
- Hyperreflexia
- Hypotension
- ECG
    - Wide QRS
    - QT prolongation > torsades de pointes
    - ST depression

Management

- Complete neuro exam
- ECG looking for long QT
- Cardiac monitoring
- Replenish magnesium
    - $MgSO_4$ 1-2 g over 30-60 min
    - Watch for toxicity
    - Can also replenish orally
    - If seizures, 2 g over 2 min
- Monitor for concomitant hypokalemia and hypocalcemia
- Watch for toxicity (diminishing reflexes)

How You Will Be Tested

- Long QT on ECG
- Physical exam findings
- Know the association with calcium, potassium, and phosphate

Hypermagnesemia

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- 60% of total body magnesium stored in bone, only 1% is extracellular
- Magnesium follows calcium and phosphate
- Can be caused by magnesium overdose (increased laxative use)
- Toxicity in eclamptic patients with high dosing
- Can see with tumor lysis and rhabdomyolysis

Clinical Findings

- Flushing
- Nausea
- Somnolence
- Respiratory depression
- Loss of deep tendon reflexes - early sign
- ECG
    - Bradycardia
    - Atrioventricular (AV) block
    - Long PR
    - ST elevations

Management

- Obtain magnesium level
- Obtain cofactor levels (potassium, calcium, phosphate levels)
- Severe
    - CaCl 100-200 mg IV over 5-10 min
    - Consider dialysis
- Mild-moderate
    - IV hydration
- Mild
    - Stop exogenous magnesium

How You Will Be Tested

- You will be given a case of iatrogenic toxicity: know the most common causes (laxative abuse, exogenous administration in the pre-eclamptic/eclamptic patient)
- Know that diminishing and absent deep tendon reflexes can be the earliest presenting sign of toxicity
- Know that dialysis can be used in severe cases

Hypophosphatemia

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- Phosphate is important in creation of adenosine triphosphate (ATP)
- Affects body at the cellular level
- Common causes
    - Renal losses
    - Diuretics
    - Hyperparathyroidism
    - Decreased gastrointestinal absorption
    - Transcellular shifts (traditionally an intracellular ion)

Clinical Findings

- Symptomatic hypophosphatemia generally <1.0 mg/dL
- Weakness
- Muscle dysfunction
- Encephalopathy/coma

Management

- Treat underlying causes and replenish loses
- Mild: replenish orally
- Severe: IV infusion
- Do not give with Ringer's lactate

How You Will Be Tested

- Know that you cannot give an IV infusion with Ringer's lactate because of precipitation
- Know the connection with hyperparathyroidism

Hyperphosphatemia

Vanessa Cardy, MD; Gita Pensa, MD; and Jessie Werner, MD

Background

- Generally defined as a serum level >5.0 mg/dL
- Causes
    - Decreased excretion (chronic kidney disease, hyperparathyroidism)
    - Increased intake (laxative abuse)
    - Cellular shifts (lysis of cells, ie, rhabdomyolysis, tumor lysis...)
- CPP (calcium phosphate product): multiply the 2 values; value >70 associated with greater risk of calcification in myocardium, vasculature, and soft tissues

Clinical Findings

- Symptoms similar to hypocalcemia
- Primary hyperparathyroidism - low phosphate
- Secondary and tertiary - high phosphate

Management

- Diuresis
- Mild to moderate
    - Oral phosphate binders
    - Dietary changes
- Severe (>14 mg/dL)
    - May need temporary dialysis

How You Will Be Tested

- Know that temporary dialysis can be used in severe cases
- Know that pathologies that cause cell lysis can present with hyperphosphatemia

Hypoglycemia

Vanessa Cardy, MD; Gita Pensa, MD; Jessie Werner, MD

Background

- Level causing symptoms varies depending on the individual; generally <70 mg/dL
- Causes
    - Antihyperglycemic medication
    - Sepsis

Clinical Findings

- Hyperadrenergic symptoms
    - Tremors
    - Sweating
    - Tachycardia
- Neuroglycopenic symptoms (<50 mg/dL)
    - Amnesia
    - Confusion
    - Seizures
    - Coma
    - Focal neurologic findings

Management

- Fingerstick glucose as soon as possible
    - Can be falsely elevated with severe anemia and hypotension
    - If low, repeat every 30 min
- Treat underlying cause
- Glucagon 1 mg intramuscular
- D50 (50% dextrose) IV 50 mL (repeat as needed) > dextrose-containing IV fluids
- Replenish orally if no altered mental status
- Consider octreotide 50-100 mg q6h if sulfonylurea use suspected
- If thiamine deficiency suspected, give thiamine 100 mg IV and glucose
- Pediatrics administration
    - <1 y: 2-5 mL/kg D10 (10% dextrose)
    - >1 y and <8 y: 2 mL/kg D25 (25% dextrose)
    - >8 y: 1 mL/kg D50 (50% dextrose)

How You Will Be Tested

- Know the common presenting symptoms and the need to look for an underlying cause
- In patients with focal neurologic findings, glucose needs to be checked immediately

Hyper & Hypoglycemia in Diabetes

Gita Pensa, MD, and Jessica Mason, MD

Background

- Factors influencing glucose control
    - Weight loss/gain
    - Exercise
    - Diet
    - Illness
- Hyperglycemia
- Hypoglycemia

Clinical Findings

- Hyperglycemia
    - Osmotic diuresis
- Hypoglycemia
    - See section on hypoglycemia

Management

- Hyperglycemia
    - Fluids
    - Treatment depends on level and signs of acidosis
    - Electrolyte management
    - Insulin if needed
- Hypoglycemia
    - Treat with dextrose or oral intake
    - Extended observation required for sulfonylurea and meglitinides

How You Will Be Tested

- Understand that sulfonylurea can cause prolonged hypoglycemia and that these patients need an extended period of observation

Hypokalemia - Diagnosis

Vanessa Cardy, MD; Stuart Swadron, MD; and Jessie Werner, MD

Background

- Decreased serum potassium level
- Can present with hypomagnesemia as well
- Transcellular shifts
- Decreased potassium intake
- Increased potassium output
    - Vomiting
    - Diuretic use
    - Renal tubular acidosis

Clinical Findings

- Symptoms
    - Central nervous system disturbance (cramps, weakness, hyporeflexia)
    - Gastrointestinal - ileus, hepatic encephalopathy
    - Cardiac dysrhythmias
    - Rhabdomyolysis
- Hypokalemic periodic paralysis - genetic condition caused by intracellular shifting of potassium in a patient with thyroid issues: ascending paralysis starting in the lower extremities
- ECG changes
    - Prominent U waves
    - Flattened T waves
    - ST depressions
    - QT prolongation
    - Bradycardia

Management

- Check and replenish magnesium as well if low
- K = 2.5-3.5
    - Oral repletion
- K < 2.5
    - IV repletion and generally admission
- See detailed section on Hypokalemia - Treatment for additional details

How You Will Be Tested

- Understand the differential diagnosis for ascending paralysis
- Know the ECG changes associated with hypokalemia

Hypokalemia - Treatment

Vanessa Cardy, MD, and Stuart Swadron, MD

Background

- See Hypokalemia - Diagnosis section
- Aim for a minimum potassium level of 3.5 mmol/L
- Can only give 10 mEq/h via peripheral access

Clinical Findings

- See section on Hypokalemia - Diagnosis

Management

- Check and replenish magnesium as well if low
- 10 mEq of potassium should raise potassium level by 0.1
- K = 2.5-3.5
    - Oral repletion with 40 mEq in ED
    - 20 mEq daily as outpatient
- K < 2.5
    - IV repletion and, generally, admission
    - Aim for K = 3.5
    - Address underlying cause
- See detailed section on Hypokalemia - Treatment for additional details

How You Will Be Tested

- Understand that higher concentrations cannot be given via a peripheral IV

See EM:RAP 2006 March - Electrolyte Emergencies

Hyperkalemia - Diagnosis

Vanessa Cardy, MD, and Stuart Swadron, MD

Background

- Increased serum potassium level
- Pseudohyperkalemia
    - Caused by hemolysis
- Other causes
    - Renal failure
    - Potassium sparing diuretics
    - Angiotensin-converting enzyme (ACE) inhibitors
    - Transcellular shift
        - Insulin deficiency
        - Crush, burns
        - Disseminated intravascular coagulation
    - Increased potassium intake
    - Massive transfusion

Clinical Findings

- ECG findings
    - Generally seen at levels <6.5 mmol/L
    - T wave peaking
    - P wave flattening
    - QRS widening
    - Sine wave pattern
    - Ventricular fibrillation
    - Heart blocks

Management

- Immediate ECG
- Treat underlying causes
- See Hyperkalemia-Treatment section for more details

How You Will Be Tested

- ECG findings

Hyperkalemia - Treatment

Vanessa Cardy, MD, and Stuart Swadron, MD

Background

- Tailored toward clinical presentation

Clinical Findings

- See section on Hyperkalemia - Diagnosis

Management

- Cardiac monitoring
- Calcium
    - Give if patient has cardiac conduction abnormalities
    - Gluconate or chloride
        - CaCl can cause sclerosis through peripheral line
        - Ca gluconate 2-4 g IV
        - CaCl 1-2 g IV (3 times more elemental calcium than calcium gluconate)
    - Consider digoxin toxicity (give digoxin-specific antibody [Fab] fragments rather than calcium)
- Insulin
    - Temporarily shifts potassium
    - Given with dextrose to prevent hypoglycemia
    - Give 5-10 units
        - Use 5 units in patients with renal failure due to prolonged effect
- $NaHCO_3$
    - If acidotic, give 50-100 mEq slowly
- Albuterol
    - 5-10 mg nebulization to cause temporary shift
- Kayexalate
    - 15-30 g orally or rectally
- Furosemide
- Consider nephrology consultation for dialysis

How You Will Be Tested

- ECG findings
- Remember that calcium is important for cardiac membrane stabilization

Hyponatremic & Seizing

Jennifer Farah, MD, and Mel Herbert, MD

Background

- Severe vs non-severe form

Clinical Findings

- Severe form
    - Coma
    - Seizures
    - Altered mental status

Management

- Hypertonic saline
    - 100 mL of 3% saline over 10 min
- Do not treat chronic hyponatremia with hypertonic saline

How You Will Be Tested

- Features of severe hyponatremia
- Osmotic demyelination syndrome with rapid correction of chronic hyponatremia

Hyponatremic & Stable

Jennifer Farah, MD, and Mel Herbert, MD

Background

- Awake, alert
- Pseudohyponatremia
    - Hyperglycemia
    - Hyperlipidemia
    - Multiple myeloma
- Hypovolemic
    - Renal insufficiency
        - Urine Na >20 due to inability to concentrate urine
    - Gastrointestinal losses
        - Urine Na <10 because kidneys trying to concentrate urine
- Hypervolemic
    - Congestive heart failure
    - Cirrhosis
    - Nephrotic syndrome
- Euvolemic
    - Endocrine disorders (low cortisol or hypothyroid)
    - Psychogenic polydipsia
    - Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
        - Consider in lung cancer patients with hyponatremia
        - Urine osmolality >150 and urine Na >20

Clinical Findings

- Hypovolemic
    - Dry mucous membrane
    - Clinical dehydration
- Hypervolemic
    - Peripheral edema
    - Pulmonary edema

Management

- Based on symptom severity
- Treat underlying cause
- Mild cases - fluid restriction
- If patient requires fluids, give normal saline
- Chronic forms - replenish slowly to avoid osmotic demyelination syndrome

How You Will Be Tested

- A clinical stem where you are asked to identify a classic cause of hyponatremia given physical exam findings

- Differentiating between presentation and management of severe vs non-severe hyponatremia

Hypernatremia

Jennifer Farah, MD, and Mel Herbert, MD

Background

- Increased serum sodium levels: Na >145 mmol/L
- Related to free water intake and balance
- Causes
    - Reduced intake of free water
    - Free water losses
        - Osmotic diuresis
        - Diabetes insipidus
            - Decreased antidiuretic hormone (ADH) production or renal sensitivity leading to kidneys losing free water

Clinical Findings

- Severe form
    - Seizure
    - Altered mental status
    - Cardiac dysrhythmias

Management

- Rate of correction = rate at which it occurred to avoid cerebral edema
    - 2-3 mEq over 4-6 h

How You Will Be Tested

- Free water deficit calculation: 0.6 × weight (kg) × [(actual sodium/140) - 1]
- Know that the dreaded consequence of rapid correction is cerebral edema

Hypothyroidism

Vanessa Cardy, MD; Gita Pensa, MD; and Mel Herbert, MD

Background

- Severe form - myxedema coma
- Insufficient production of thyroid hormone
- Can occur after a period of thyrotoxicosis
- Autoimmune - Hashimoto thyroiditis
- Lithium or amiodarone ingestion
- Iatrogenic from hyperthyroidism treatment
- Status post radiation or thyroidectomy
- Secondary causes
    - Tumors
    - Sheehan syndrome - postpartum hemorrhage into pituitary gland

Clinical Findings

- Fatigue and lethargy
- Depression
- Weight gain
- Cold intolerance
- Menstrual irregularities
- Constipation
- Peripheral edema
- Coarse skin
- Myxedema coma
    - Hypothermia
    - Altered mental status
    - Non-pitting edema (pretibial myxedema)
    - Macroglossia
    - Hypertension
    - Bradycardia
    - Slow relaxation phase of reflexes
    - Respiratory failure

Management

- Look for vital sign abnormalities
- Physical exam, paying attention to mental status and skin
- Labs
    - High thyroid-stimulating hormone (TSH), low free thyroxine (T4)
    - Secondary hypothyroidism can cause low TSH
    - Cortisol level
- Uncomplicated hypothyroidism
    - Oral levothyroxine at 50 µg daily and schedule outpatient follow-up

- Myxedema coma
    - IV levothyroxine
    - Correct other metabolic derangements
    - Hydrocortisone 100 mg IV
    - Anticipate difficult airway given macroglossia and edema

How You Will Be Tested

- Presentation of myxedema coma, making the diagnosis of hypothyroidism

Hyperthyroidism

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- Thyrotoxicosis - excess circulating thyroid hormone
    - Excess production
    - Excess release
    - Iatrogenic
- Graves' disease
    - Most common cause in regions with adequate iodine intake
    - Increased thyroid-stimulating antibodies
- Toxic adenoma
    - Most common cause in iodine-deficient diets
    - Direct excess production of thyroid hormone
- Thyroiditis
    - Inflammatory production of thyroglobulin and release of thyroid hormone
    - Can progress into hypothyroidism before recovery
    - Subacute thyroiditis - painful thyroid
        - Post-viral
        - Medications (iodine, lithium, chemotherapy, amiodarone)
        - Infection
- Thyroid storm - Most severe form

Clinical Findings

- Tremor
- Tachycardia (can be blunted in an elderly patient)
    - Atrial fibrillation
- Hyperthermia
- Hair/nail changes
- Ophthalmopathy (Graves')
- Goiter or nodules
- Painful thyroid in subacute thyroiditis
- Gastrointestinal distress

Management

- Treat fever
- Consider glucose and thiamine
- Check thyroid panel
- Check complete blood count, electrolytes, liver function tests
- Thyroid storm
    - Consider underlying infectious cause

How You Will Be Tested

- Know the different etiologies of hyperthyroidism and be able to differentiate between them

See Crunch Time - Endocrine - Thyroid Storm

Thyroid Storm

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- Extreme form of hyperthyroidism
- In the elderly, can see apathetic thyrotoxicosis
- High mortality rate likely secondary too cardiovascular collapse or central nervous system dysfunction
- Triggered by any stressor with underlying hyperthyroidism
- Exogenous thyroid hormone
- History will be of great use

Clinical Findings

- Sympathetic stimulation
    - Confusion
    - Seizure
    - Coma
    - Tachydysrhythmia
    - Gastrointestinal symptoms
    - Hyperthermia

Management

- Thorough examination
- Low thyroid-stimulating hormone (TSH)
- Elevated triiodothyronine (T3) and thyroxine (T4)
- With secondary hyperthyroidism, can see elevated TSH as well
- Treatment
    - Supportive care
    - Treat underlying trigger
    - *In general treat with 1) Beta blocker then 2) Antithyroid medication then 3) Iodine*
    - Block peripheral thyroid hormone effect: Propranolol 1-2 mg IV q10min as needed
    - Block thyroid hormone production: Propylthiouracil (PTU) (600-100 mg orally)
    - Inhibit thyroid hormone release: Iodine (wait 1 h after PTU)
    - Inhibit conversion of T4 to T3: Dexamethasone (2 mg IV q6h)
- Endocrine consult

How You Will Be Tested

- Order of medications to give in thyroid storm
- You will be asked to identify the pathology of thyroid storm and be given a vignette of a patient with underlying hyperthyroidism undergoing a stressor

Thyroid Nodules & Tumors

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- Incidentally found on imaging
- 95% of nodules are not malignant
- Can present with hyperthyroidism if malignant
- 4 main types of thyroid cancer:
    - Papillary
    - Follicular
    - Medullary
    - Anaplastic

Clinical Findings

- Airway compromise due to mass effect
- Gland asymmetry
- Anterior neck swelling
- Sequelae of hyperthyroidism

Management

- Thyroid ultrasound if normal thyroid-stimulating hormone (TSH)/thyroxine (T4) and any of the following
    - Non-toxic goiter
    - Gland asymmetry
    - Palpable nodules
- Thyroid radioiodine scan if:
    - Thyroid nodules and low TSH with high T4
- Fine needle aspiration/biopsy if concerning features
- Ear, nose, and throat/oncologic referral

How You Will Be Tested

- Anaplastic carcinoma is most lethal
- Signs and sequelae of a thyroid nodule or goiter

Pituitary Tumors

Vanessa Cardy, MD, and Gita Pensa, MD

Background

- Microadenoma <1 cm
- Macroadenoma >1 cm
- Generally benign but can have mass effect or apoplexy
- Symptoms with excess or deficiency of hormones

Clinical Findings

- Headache
- Bitemporal hemianopsia (mass compressing optic chiasm)
- Cerebrospinal fluid rhinorrhea
- Hyper- or hypothyroidism
- Hypogonadism
- Acromegaly, gigantism, or short standard
- Galactorrhea (prolactinoma)

Management

- Hormone-specific tests
- Neuroimaging
- Endocrinology/neurosurgery consults
- CT head > MRI

How You Will Be Tested

- Galactorrhea associated with prolactinoma
- Bitemporal hemianopsia

Malabsorption

Gita Pensa, MD, and Mel Herbert, MD

Background

- Related to inadequate digestion, absorption, or transportation of nutrients
- Can be global or specific to certain nutrients

Clinical Findings

- Global
    - Diffuse mucosal disease
    - Anatomic resections
    - Pancreatic insufficiency or chronic pancreatitis
    - Inflammatory bowel disease
    - Celiac disease
    - Fat malabsorption
        - Foul-smelling, greasy diarrhea
        - Look for fat-soluble vitamin deficiencies
- Isolated
    - Classically B_{12} deficiency in pernicious anemia
    - Terminal ileum dysfunction in Crohn's disease

Management

- Supplementation of appropriate micro- and macronutrients

How You Will Be Tested

- A, D, E, and K are fat-soluble vitamins
- B_{12} deficiency associated with pernicious anemia

Thiamine Deficiency

Gita Pensa, MD, and Mel Herbert, MD

Background

- Classically associated with alcoholics

Clinical Findings

- Wernicke-Korsakoff syndrome
    - Wernicke's - confused alcoholic
    - Korsakoff syndrome - word salad
- Beri-beri
    - Polyneuropathy
    - High-output heart failure

Management

- Replenish thiamine before dextrose

How You Will Be Tested

- Components of Wernicke-Korsakoff syndrome

Niacin Deficiency

Gita Pensa, MD, and Mel Herbert, MD

Background

- Pellagra

Clinical Findings

- Pellagra
    - Photosensitive pigmented dermatitis
    - Diarrhea
    - Dementia

Management

- Supplementation of niacin

How You Will Be Tested

- Know the 3 Ds of pellagra: dermatitis, diarrhea, and dementia

B_6 Deficiency (Pyridoxine)

Gita Pensa, MD, and Mel Herbert, MD

Background

- Pyridoxine

Clinical Findings

- Seizure with severe deficiency
- Neuropathy
- Anemia

Management

- Supplementation of pyridoxine

How You Will Be Tested

- Isoniazid toxicity causes B_6 deficiency

Scurvy

Gita Pensa, MD, and Mel Herbert, MD

Background

- Vitamin C (ascorbic acid) deficiency
- Malnourished
- Malabsorption
- Alcoholics
- Lack of fresh fruits and vegetables

Clinical Findings

- Bruising
- Connective tissue problems

Management

- Supplementation of vitamin C

How You Will Be Tested

- Know the symptoms of scurvy

Vitamin B_{12} & Folate Deficiency

Gita Pensa, MD, and Mel Herbert, MD

Background

- Patients following bariatric surgery
- Vegetarians and vegans
- Alcoholics

Clinical Findings

- Macrocytic anemia
- Hypersegmented neutrophils

Management

- Supplementation of respective vitamin

How You Will Be Tested

- Case stem: patient with history of bariatric surgery presenting with rash and neurologic findings

Fat-Soluble Vitamin Deficiencies

Gita Pensa, MD, and Mel Herbert, MD

Background

- Vitamins A, D, E, and K
- Can overdose as they can accumulate in the body

Clinical Findings

- Vitamin K deficiency - coagulopathy
- Vitamin D deficiency - bony breakdown

Management

- Supplementation of respective vitamin

How You Will Be Tested

- Vitamin D: In kids, rickets; in adults, osteomalacia

Wernicke-Korsakoff Syndrome

Vanessa Cardy, MD, and Mel Herbert, MD

Background

- Thiamine deficiency
- Wernicke's encephalopathy
- Korsakoff syndrome

Clinical Findings

- Wernicke's encephalopathy
    - Ataxia
    - Poor coordination
    - Nystagmus
    - Ophthalmoplegia
- Korsakoff syndrome
    - Memory loss
    - Apathy
    - Confabulation

Management

- Supplementation of thiamine
- Give magnesium with thiamine
- Screen for other effects of chronic alcoholism

How You Will Be Tested

- Thiamine before glucose
- Case stem with confused alcoholic; treatment is high-dose thiamine