

Week 11:

- Diabetes insipidus → deficit of ADH
 - Lab findings
 - Urine chemistry (think dilute)
 - Decrease specific gravity
 - Decreased osmolality
 - Serum chemistry (think concentrated)
 - Increased osmolality
 - Increased sodium
 - Increased potassium
 - Diagnostic procedures: water deprivation test (ADH stimulation test & vasopressin test)
 - Expected findings
 - Polyuria (abrupt onset of excessive urination → 4-30L/day)
 - Polydipsia
 - Nocturia
 - Fatigue
 - Dehydration
 - Pharmacologic treatment = desmopressin
 - Client Education:
 - Lifelong medication
 - Notify PCP if weight gain > 0.9kg in 24 hours; if HA or confusion occur
 - Nursing Considerations:
 - Monitor VS and urine output
 - Use cautiously if hx of CAD d/t vasoconstriction
 - Monitor for HA, confusion, or other indications of water intoxication
- Cushing's → excess cortisol
 - Disease → tumor in pituitary gland releasing ACTH increasing cortisol
 - Syndrome → long-term glucocorticoid use from asthma or RA
 - Manifestations
 - Weakness/fatigue
 - Altered emotional state (irritability, depression)
 - Decreased libido
 - Sleep disturbances
 - Back and joint pain
 - Physical assessment findings
 - Classic picture → central-type obesity, with a fatty "buffalo hump" in the neck and supraclavicular areas, a heavy trunk, and relatively thin extremities

- Evidence of decreased immune function and decreased inflammatory response
 - Thin, fragile skin
 - Bruising/petechiae
 - Tachycardia
 - Gastric ulcers d/t over secretion of hydrochloric acid
 - Weight gain and increased appetite
 - Interventions
 - Labs: elevated plasma cortisol levels, plasma ACTH levels
 - XR, MRI, CT for potential lesion identification
 - Therapeutic: chemo, radiation, hypophysectomy, adrenalectomy
 - Medications: tapering glucocorticoids, Ketoconazole (antifungal inhibiting corticosteroid synthesis), Mitotane (selective destruction of adrenocortical cells), Hydrocortisone (replacement therapy)
 - RN considerations
 - Monitor I&O, daily wt, CBC WBC
 - Assess for hypervolemia
 - Maintain safe environment to minimize pathologic fracture and skin trauma
 - Reposition q2h and encourage physical activity w/in pt limitations
 - Frequent hand hygiene w/ meticulous skin care
 - Monitor for and protect against skin break down and infection
 - Surgical asepsis w/ dressing changes and invasive procedures
- Addison's → deficit of cortisol
 - Causes
 - Damage or dysfunction of adrenal cortex → diminished mineralocorticoid and glucocorticoid production resulting in decreased aldosterone and cortisol
 - TB and histoplasmosis are the most common infections that destroy adrenal gland tissue
 - Therapeutic use of corticosteroids is the most common cause of adrenocortical insufficiency
 - Surgical removal or infection of adrenal glands
 - Inadequate secretion of ACTH from the pituitary gland d/t decrease stimulation of the adrenal cortex
 - Manifestations
 - N/V/D/C/abdominal cramping
 - Dizziness w/ orthostatic hypotension
 - Hyperpigmentation
 - Weight loss/dehydration
 - Craving for salt
 - Hyponatremia/hypoglycemia
 - hyperkalemia/hypercalcemia
- SIADH → excess ADH

- Manifestations
 - Early:
 - HA
 - Weakness
 - Anorexia
 - Muscle cramps
 - Weight gain (w/o edema d/t water, not sodium, retention)
 - As serum Na⁺ decreases:
 - N/V/D
 - Personality changes
 - Hostility
 - Sluggish DTRs
 - Oliguria w/ dark yellow concentrated urine
- Interventions
 - RN considerations:
 - Restrict oral fluids
 - Monitor I&O and daily wt; urine and blood chemistry
 - Monitor VS → HTN, tachycardia, hypothermia
 - Auscultate lung sounds for pulmonary edema
 - Report altered mental status
 - Reduce environmental stimuli
 - Medications: tetracycline derivative (correct fluid/electrolyte imbalances by stimulating urine flow), vasopressin antagonist, loop diuretics, hypertonic sodium IV fluids
- Pheochromocytoma → excess catecholamines (epinephrine)
 - Rare condition caused by a tumor in the adrenal medulla
 - Avoid palpation of abdomen d/t risk of severe HTN
- Manifestations
 - Severe episodic hypertension accompanied by a severe, pounding HA, tachycardia w/ palpitations, profuse diaphoresis, and unexplained abdominal or CP
 - RN: monitor BP if “attack” is occurring
 - Classic triad → severe pounding HA, tachycardia, profuse sweating
- Thyroid storm
 - Rare, life threatening condition characterized by severe clinical manifestations of thyrotoxicosis
- Causes
 - Uncontrolled hyperthyroidism occurring most often with Grave’s disease
 - Acute event → infection, trauma, emotional stress, DKA, digitalis toxicity
 - Following a surgical procedure or a thyroidectomy as a result of manipulation of the gland during surgery
- Manifestations

- Hyperthermia/hyperpyrexia (104-106F)
 - Delirium/agitation
 - Vomiting/abdominal pain
 - HTN/chest pain
 - Tachydysrhythmias/palpitations
- Pharmacologic treatment
 - Beta blockers
 - Thionamide
 - Glucocorticoids
 - An iodine solution
 - An iodinated radiocontrast agent
- Nursing interventions
 - Maintain a patent airway
 - Provide continuous cardiac monitor for dysrhythmias
 - Administer acetaminophen to decrease temperature
 - Salicylate antipyretics (ASA) are contraindicated because they release thyroxine from protein-binding sites and increase free thyroxine levels
- Myxedema
 - Coma that is rare but serious d/o results from persistently low thyroid production
 - Life-threatening condition that occurs when hypothyroidism is untreated or when a stressor (EX: acute illness, surgery, chemo, d/c thyroid replacement therapy, or use of sedative/opioid) affects the client who has hypothyroidism
- Manifestations
 - Respiratory failure/hypoventilation
 - Hypotension
 - Bradycardia/dysrhythmia
 - Hypothermia
 - Hyponatremia/Hypoglycemia
 - Coma
- Nursing interventions
 - Maintain airway patency
 - Initiate aspiration precautions
 - Administer IV fluids as prescribed

Week 12:

- Diabetes mellitus
 - Diagnostic labs and values pertinent to each (2 findings on separate days)
 - Fasting blood glucose >126 mg/dL (fasting= no caloric intake x 8hrs)
 - Manifestations of diabetes plus casual blood glucose concentration >200 mg/dL (w/o regard to time since last meal)
 - 2-hr glucose >200 mg/dL w/ oral glucose tolerance test

- Glycosylated hemoglobin (A1C) >6.5% measures 8-12 weeks
 - Diabetics A1C: <7
 - None diabetics A1C: <5
- Hypoglycemia
 - Manifestations
 - Mild: hunger, nervousness, palpitations, sweating, tachycardia, tremor
 - Moderate: confusion, double vision, drowsiness, emotional changes, H, impaired coordination, inability to concentrate, irrational or combative behavior, lightheadedness, numbness of the lips and tongues, slurred speech
 - Severe: difficulty arousing, disoriented behavior, loss of consciousness, seizures
 - Interventions
 - Checks blood glucose level
 - For a conscious pt administer simple carbohydrate to treat hypoglycemia
 - Commercially prepared glucose tablets
 - 6-10 life savers or hard candy
 - 4 tsp of sugar
 - 4 sugar cubes
 - 1 tbsp of honey or syrup
 - ½ cup of fruit juice or regular (non diet) soft drink
 - 8 oz of low fat milk
 - 6 saltine crackers
 - 3 graham crackers
 - Recheck blood sugars after 15 mins of administering carbohydrates and give more if it is still low
 - If the client is unconscious administer glucagon subQ or IM. place the client into a lateral position to prevent aspiration
 - Should administer IV injection of 25-50mL of 50% dextrose in water
 - DO NOT GIVE AN UNRESPONSIVE PT ORAL FOODS
- DKA can occur after stress of the body such as an infection or trauma
 - Manifestations
 - Polyuria, polydipsia, and marked fatigue
 - Blurred vision, weakness, and headache
 - Orthostatic hypotension from loss of fluids
 - Frank hypotension with a weak, rapid pulse
 - GI symptoms such as anorexia, N/V, and abdominal pain
 - Acetone breath (a fruity odor)
 - Hyperventilation (with very deep, but not labored respirations) may occur- kussmaul respirations
 - Pt may be alert, lethargic, or comatose
 - Lab findings
 - Blood glucose levels 300-1000

- Low serum bicarbonate, low pH; low PCO₂, reflects respiratory compensation (Kussmaul's respirations)
 - Ketones bodies in blood and urine
 - Electrolytes vary according to degree of dehydration; increase in creatinine, Hct, BUN
 - Major electrolyte concern is POTASSIUM
 - Treatment
 - Rehydration- FLUID REPLACEMENT
 - Restoring electrolytes
 - Reverse acidosis
 - Initial fluid of choice is 0.9% NS- rate of infusion dependent on clinical state of patient
 - If hypernatremic- 0.45% saline in order to provide electrolyte free water
 - When the BG reaches 200-300 or less- changes to D5W to prevent decline in blood glucose level and so that insulin can be contributed to achieve ketones clearance
 - Insulin administration
 - Only regular insulin can be administered IV
 - Infused at a slow continuous rate to help reverse acidosis (5 units/hr)
 - Hourly blood glucose values must be measured
 - Insulin must be infused continuously until subQ administration of insulin can be resumed; any interruption in administration may result in the reaccumulation of ketone bodies and worsening conditions
- HHS- blood glucose levels 600-1200. Ketosis and acidosis does not generally occur in HHS
 - Treatment
 - Fluid replacement
 - Listen to lung sounds and monitor I&O's
 - Correction of electrolyte imbalances
 - Insulin administration
 - Insulin is usually administered at a continuous low rate to treat hyperglycemia, and replacement IV fluids with dextrose are administered after glucose level has decreased to the range of 250-300 mg/dL
- Pancreatitis
 - Manifestations
 - Sudden onset of severe, boring pain (goes through the body)
 - Epigastric, radiating back, left flank, or left shoulder
 - Worse when lying down
 - Pain unrelieved somewhat by fetal position or sitting upright, bending forward
 - N/V, weight loss
 - Turner's sign —> ecchymosis on the flanks
 - Bluish-gray periumbilical discoloration —> Cullen's sign
 - Generalized jaundice

- Absent or decreased bowel sounds
 - Warm, moist skin, fruit breath
 - Ascites
 - Lab findings
 - Increased amylase & lipase
 - Increased wbc
 - Increased serum liver enzymes (ALT, AST) & bilirubin
 - Pain-relief interventions
 - Opioid analgesics (morphine, fentanyl)
- EGD
 - Client education pre-procedure
 - No eating 6-12 hours prior to procedure
 - Educate before because they will receive twilight sedation
 - Expect sore throat, light blood tinged sputum after EGD
 - Nursing interventions
 - Informed consent prior
 - Assess for gag reflex after
- GI bleeds
 - Priority assessment
 - Vital Signs—> assess for hypovolemia/shock (hypotension, tachycardia)
- AKI
 - Causes
 - Prerenal acute kidney injury
 - Renal vascular obstruction
 - Shock
 - Decreased cardiac output causing decreased renal perfusion
 - Sepsis
 - Hypovolemia
 - Peripheral vascular resistance
 - Use of aspirin, ibuprofen, or NSAIDs
 - Liver failure
 - Intrarenal acute kidney failure
 - Physical injury: trauma
 - Hypoxic injury: renal artery or vein stenosis or thrombosis
 - Chemical injury: acute nephrotoxins (antibiotics, contrast dye, heavy metals, blood transfusion reaction, alcohol, cocaine)
 - Immunologic injury: infection, vasculitis, acute glomerulonephritis
 - Lab findings
 - Serum creatinine gradually increases 1 to 2 mg/dL every 24 to 48 hours, or 1 to 6 mg/dL in 1 week or less
 - BUN can be increased to 80 to 100 mg/dL within 1 week
 - Urine specific gravity varies in postrenal type, can be elevated up to 1.030 in prerenal type or diluted as low as 1,000 in intrarenal type

- Sodium can be decreased in prerenal azotemia or increased intrarenal azotemia
 - Hematocrit is decreased
 - Urinalysis presence of sediment
 - Metabolic acidosis
- CKD
 - Dietary restrictions with examples of each
 - Instruct the client to monitor the daily intake of carbohydrates, proteins, sodium, and potassium, according to the provider's prescription
 - Instruct the client to avoid antacids containing magnesium
 - Drink at least 2 liters of water daily
 - Stop smoking and limit alcohol intake
 - Lab findings
 - Urinalysis: hematuria, proteinuria, and decrease in specific gravity
 - Serum creatinine: gradual increases over months to years for CKD exceeding 4 mg/dL. Can increase to 15 to 30 mg/dL
 - BUN increased with elevated serum creatinine
 - Decreased sodium and calcium; increased potassium, phosphorus, and magnesium
 - CBC: decreased hemoglobin and hematocrit from anemia secondary to the loss of erythropoietin in CKD
 - Nursing interventions
 - Report and monitor irregular findings
 - Assess and monitor vascular access or peritoneal dialysis insertion site
 - Obtain a detailed medication and herb history to determine the client's risk for continued kidney injury
 - Control protein intake based on the client's stage of CKD and type of dialysis prescribed
 - Restrict dietary sodium, potassium, phosphorus, and magnesium
 - Provide a diet that is high carbohydrates and moderate in fat
 - Restrict intake of fluids (based on urinary output)
 - Adhere to meticulous cleaning of areas on skin not intact and access sites to control infections
 - Balance the client's activities and rest
 - Prepare the client for hemodialysis, peritoneal dialysis, and hemofiltration if indicated
 - Provide skin care in order to increase comfort and prevent breakdown
 - Provide emotional support to the client and family
 - Encourage the client to ask questions and discuss fears
 - Administer medications as prescribed
- Peritoneal dialysis
 - What is it, how does the system work

- Involves instillation of hypertonic solution into the peritoneal cavity and subsequent dwell times, drain the dialysate solution that includes the waste productions, the peritoneum serves as the filtration membrane
 - Client should have intact peritoneal membrane without adhesions from infection or multiple surgeries
- ESRD
 - Manifestations
 - Neurologic
 - Asterixis, behavior changes, burning of soles of feet, confusion, disorientation, inability to concentrate, restlessness of legs, seizures, tremors, weakness and fatigue
 - Integumentary
 - Coarse, thinning hair, dry and flaky skin, ecchymosis, gray-bronze skin color, pruritis, purpura, thin, brittle nails
 - Cardiovascular
 - Engorged neck veins, hyperkalemia, hyperlipidemia, hypertension, pericardial friction rub, pericardial tamponade, pericarditis, periorbital edema, pitting edema
 - Pulmonary
 - Crackles, depressed cough reflex, kussmaul-type respirations, pleuritic pain, shortness of breath, tachypnea, thick and tenacious sputum, uremic pneumonitis
 - Gastrointestinal
 - Ammonia odor to breath, anorexia, nausea, vomiting, bleeding from gastrointestinal tract, constipation or diarrhea, hiccups, metallic taste, mouth ulcerations and bleeding
 - Hematologic
 - Anemia, thrombocytopenia
 - Reproductive
 - Amenorrhea, decreased libido, infertility, testicular atrophy
 - Musculoskeletal
 - Bone fractures, bone pain, foot drop, loss of muscle strength, muscle cramps, renal osteodystrophy
- Kidney biopsy
 - Nursing interventions
 - Client receive sedation and ongoing monitoring
 - Pre-procedure
 - Obtain informed consent
 - Obtain urine specimen
 - Review coagulation studies
 - NPO for 4-6 hours
 - Post-procedure
 - Monitor VS following sedation
 - Assess dressings and UO (hematuria)

- Review Hgb and Hct values
 - Administer prn pain medications
- Kidney transplants
 - Contraindications
 - Age <2
 - Age >70
 - Advanced, untreatable cardiac disease
 - Chemical dependency
 - Chronic infections or system diseases
 - Coagulopathies and certain immune diseases
 - Morbid obesity
 - DM
 - COPD
 - Untreated GI disease, such as peptic ulcer disease
 - Organ rejection manifestations
 - Hyperacute: occurs within 48 hours after surgery
 - Fever, hypotension, pain at the transplant site
 - Acute: occurs 1 week to 2 years after surgery
 - Oliguria, anuria, low-grade fever, hypotension, tenderness over the transplanted kidney, lethargy, azotemia, and fluid retention
 - Chronic: occurs gradually over months to years
 - Gradual return of azotemia, fluid retention, electrolyte imbalance, and fatigue
- Dialysis types
 - Hemodialysis: a procedure that circulates the patient's blood through an artificial kidney to remove waste products and excess fluid
 - Peritoneal dialysis: a procedure that uses the patient's peritoneal membrane (the lining of the peritoneal cavity) as the semipermeable membrane to exchange fluid and solutes