**Study Guide**

**Exam 2**

-Liver is the largest gland in the body

-Makes bile (breakdown fat)

-Stores cholesterol and glycogen

-excretes meds

-metabolism

-located in RUQ above rib cage and should not be palpable in the abdomen.

-below liver is the gallbladder which stores the bile that the liver produces

**Functions**

-stores glucose as glycogen, blood glucose regulation

-makes albumin which keeps oncotic pressure

-blood clotting factors

-lipoproteins: transport cholesterol to and from liver and body

-storage of ADEK vitamins

-drug metabolism

**Hepatic Cirrhosis**

-liver tissue replaced by scar tissue

-causes: alcohol and infection like Hepatitis (specifically Hep B and C)

\*Chronic inflammation changes normal tissue to scar tissue

**S/s:**

-Respiratory distress

-abdominal pain

-constipation or diarrhea

-possible esophageal, stomach, and anal varices which can dilate and rupture

-dyspnea

-weight loss

-ascites

-splenomegaly

**Treatment**

-rest

-improve nutrition: normal protein intake if there is no hepatic encephalopathy

-skincare

-prevention of bleeding: no coughing, sneezing, straining

-administer lactulose to help excrete ammonia faster

-diuretics

-paracentesis to diagnose and decrease volume of fluid in abdominal cavity

\*Void before procedure

\*Monitor fluid removal should be no more than 3000mL

Treatment for bleeding

-vasopressin: constricts splanchnic arterial bed

-balloon tamponade: sengstaken-blakemore tube

**Hepatitis: inflammation of the cover of the liver**

-viral hep, cytomegalovirus, Epstein-Barr virus, herpes, coxsackievirus, rubella

-30% hep B asymptomatic

-80% hep C asymptomatic

-S/s: jaundice, clay colored feces, dark urine, splenomegaly

-LABS: AST, ALT elevated up to 100X’s.

**Test**: is it chronic? How is it transmitted? How can it be prevented?

**A – same transmission as E**

\*Common in developing countries where water and food don’t have the same quality standards

\*Still happens in developed countries

\*Transmitted: Fecal/oral

\*When infected they don’t have symptoms

\*if jaundiced they are not infectious

\*Not chronic. Once infected you are cured for life.

\*To prevent: Wash hands properly.

**B – same transmission as C and D**

\*Can become chronic

\*Incidence decreased because of vaccine

\*Transmission: IV drug used, perinatally by mothers to infant, sexually transmitted, can live on dry surface for 7 days, kissing

\*Cure: body must be develop anti HBs IgG

\*Without treatment they will develop end stage liver failure or cancer, become chronic carrier which transmits to others.

**C – same transmission as B and C**

\*Can become chronic

\*30-40% of people with HIV have Hep C

\*Transmitted most commonly during IV drug use, sex, hemodialysis, occupational exposure, perinatal.

\*Never develop antibodies/immunity. Can be reinfected if behavior is not rectified.

**D – Same transmission as B and D**

\*Cannot infect body without Hep B being present

\*transmitted percutaneously

\*Fulminant hepatitis. Hep B and D coinfection. Can cause acute liver failure. Most severe hep.

**E – same transmission as A**

\*Transmitted: Fecal/oral

G -

\*Parenterally transmitted.

\*Does not appear to cause liver damage

**Endocrine**

Pituitary

**Thyroid**: surrounds portion of trachea

-2 lobes with isthmus

-TSH: thyroid stimulating hormone. Released from anterior pituitary gland.

\*turns on iodine pumps

-produces T3, T4, and calcitonin

-controls cellular metabolic activity

**Parathyroid**

-regulates calcium and phosphorus metabolism

-PTH: moves Ca+ from bones to blood

**Hyperparathyroidism**

-caused by overproduction of parathormone: causes kidney stones containing Ca+ and bone decalcification

S/s: apathy, fatigue, vomiting, constipation, HTN, dysrhythmias, irritability, increased peptic ulcers and pancreatitis

-Hypercalcemia crisis: neuro, cardiac, and kidney symptoms – rapid rehydration with isotonic solutions with calcitonin to maintain urine output of 100-150 p/h. Monitor for fluid overload.

**Hypoparathyroidism**

Cause: destruction of parathyroid, Vit D deficiency

-Hyperphosphatemia and hypocalcemia

S/s: tetany from hypocalcemia, numbness, tingling, and cramps in extremities

Treatment: calcium gluconate, sedative, thiazide diuretic, Vit D, tracheostomy kit at bedside, high Ca+ low P-

\*If patient has heart condition and takes digitalis administer Ca+ cautiously. They potentiate each other and can cause fatal dysrhythmias. Continuous cardiac monitoring required.

Adrenal

**Complications of Trans-sphenoidal hypophysectomy**

-CSF leak

-Increased ICP

-Meningitis

**Hypothyroidism risks, treatment, S/s, and nursing interventions**

-most common cause is Hashimoto’s disease

-also caused by PT’s treating hyperthyroidism with radioiodine, anti-thyroid meds or thyroidectomy

**Risks:** female, 30-60 y/o, treatment for hyperthyroidism

**Labs**: decreased T3 and T4 with increased TSH, increased cholesterol

**S/s**: extreme fatigue, hair loss, brittle nails, dry skin, numbness and tingling in fingers, cold intolerance, constipation, weight gain, pallor, brittle nails, bradycardia, hypotension, dry, flaky skin, hoarse voice, MI, masklike expression.

**Treatment**: Levothyroxine, takes 6 months for full effect, take in the morning, on an empty stomach with a full glass of water, increase AC, warm blankets, I&O, daily weight, increase fiber and fluid

**Sign and Symptoms Interventions for myxedema coma – extreme hypothyroidism**

-usually occurs in undiagnosed hypothyroidism

**S/s:** depression, altered mental status, lethargy, respiratory depression, coma, hyponatremia hypoglycemia, hypotension, bradycardia, hypothermia

**Interventions:** IV levothyroxine, mechanical ventilation, replace fluids with 0.9% NaCl IV

**Hyperthyroidism S/S medical treatment and medications side effects**

-overproduction of T3 and T4 = increased metabolic rate

-Grave’s disease is most common cause

**Cause**: shock, stress, or an infection

**S/s**: hyperexcitable, irritable, apprehensive, jittery, palpitations, tachycardia – may lead to CHF, HTN, skis is warm, soft, and moist, heat intolerance, hyperhidrosis, tremor, exophthalmos, weight loss, amenorrhea, diarrhea, dyspnea

**Complication**: thyroid storm, cardiac issues: note the presence of dyspnea, crackles, peripheral edema, JVF, and S3 heart sound

**Labs**: increased T3, T4 and decreased TSH

**Treatment**

1. Radioisotope Iodine 131 Therapy – destroys overactive thyroid cells, most PT’s only need 1 dose, observe for signs of thyroid storm, methimazole administered 4-6 weeks prior to administration, monitor until euthyroid state is achieved. Methimazole is stopped 3 days before radioactive iodine treatment. Restarted 3 days after and tapered off over 4-6 weeks. 2-3 months before therapeutic effect. Levothyroxine started 4-18 weeks after methimazole has been stopped.
2. Antithyroid agents: block utilization of iodine which prevents the synthesis of T3 and T4.
   1. Propylthiouracil (PTU): use during 1st semester of pregnancy and then switched to MMI. Hepatoxic.
   2. Methimazole: preferred drug but it is teratogenic
   3. Take in the morning on an empty stomach with a full glass of water
   4. Symptom relief takes several weeks because previously formed T3 and T4 is still in the body.
3. Beta blockers - propranolol to deal with tachycardia and HTN.
4. Sub total thyroidectomy: stop taking blood thinks a few weeks before surgery
5. Corticosteroids – hydrocortisone to treat shock.

**Antithyroid medications education**

-take in the morning, on an empty stomach, followed by a full glass of water

-do not stop abruptly

-take at the same time everyday to keep therapeutic level

-labs every 2 weeks until titrated appropriately

-takes 6 months after proper dose achieved for full effect

**Complications and nursing precautions post sub-total thyroidectomy**

**Thyroid storm:** caused by uncontrolled hyperthyroidism. Can happen during thyroidectomy b/c of manipulation of thyroid gland. S/s: hyperthermia, HTN, delirium, vomiting, ab pain, tachycardia, chest pain, dyspnea, palpitations, high fever, altered mental status.

**Airway obstruction:** hemorrhage, tracheal collapse, mucus accumulation, laryngeal edema, and vocal cord paralysis. Monitor for stridor and restlessness. Keep tracheostomy tray and suction kit at bedside. Bed should be in high fowlers. Humidified air.

**Addison and Cushing’s, dietary recommendations, diagnosis labs**

**Addison’s diet:** high sodium, carbs, and protein. Low K+. Avoid alcohol and caffeine

**Cushing’s diet:** high protein calcium and vit D to minimize muscle wasting and osteoporosis. Low Na+, low calorie, low carb.

**Addisonian crisis s/s and management**

-adrenal insufficiency: adrenal glands are damaged and cannot produce enough cortical hormones.

**Causes**: TB or other infection, surgical removal, atrophy, use of corticosteroids, sudden stop of hormone therapy

**S/s:** shock, hypotension, tachypnea, pallor, extreme weakness, rapid and weak pulse.

**Treatment**: restore fluid balance, high sodium foods, hormone replacement, avoid stress, prevent infection,

**Addison’s disease treatment and evaluation**

**Treatment**: hydrocortisone IV for shock, D5 in 0.9% NaCl for hypoglycemia, vasopressors for hypertension, antibiotics for infection

**S/s:** **everything low except K+ and WBC,** muscle weakness, anorexia, GI symptoms, fatigue, emaciation, hypotension, hypocalcemia, hyponatremia, hypoglycemia, hyperkalemia, depression, apathy, confusion

**Cushing’s s/s**

-caused by corticosteroid use

S/s: central obesity, buffalo hump, thin extremities, ecchymoses, disturbed sleep, muscle wasting, osteoporosis, kyphosis, HTN, heart failure, hyperglycemia, moonfaced, slow healing, hirsutism, voice deepens,

**Prevention of crisis in pheochromocytoma**

-don’t press the belly

-no stress

-prevent trauma

**Adrenal Cortex**

– produces glucocorticoids – cortisol: affects glucose, protein, fat metabolism

-mineralocorticoids – aldosterone, sex hormones – androgens: increases Na absorption and K+ excretion in the kidneys

**Pheochromocytoma**

-benign tumor that originates in the adrenal medulla

-strong family history ties

S/s: headache, diaphoresis, palpitations, HTN (as high as 250/150), anxious, tremulous, weak, headache, vertigo, blurred vision, tinnitus, dyspnea, polyuria, n/v, diarrhea, ab pain, feeling of impending doom. Five H’s: HTN, headache, hyperhidrosis, hyperglycemia, hypermetabolism

Complications: dysrhythmias, dissecting aneurysm, stroke, AKI

**Addison’s**

-Everything low except K+ and WBC

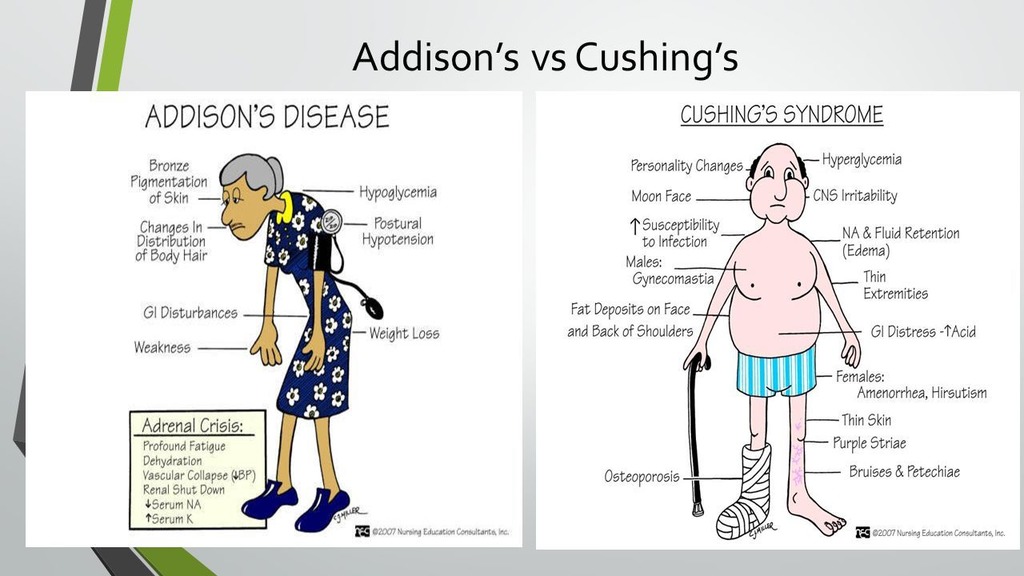
-crisis caused by cold, infection, shock, overexertion, dehydration, stress

-weight loss, depressed, low energy, cold intolerance, hair loss, low blood pressure, hypoglycemia, salt craving

**Cushing’s**

-Everything high except K+

-weight gain, HTN, hyperglycemia, truncal obesity, moon face, buffalo hump, hirsutism, purple striae, slow wound healing, osteoporosis



**Diabetes insipidus s/s and labs**

-deficiency of ADH – vasopressin

-ADH is secreted by the pituitary gland so if it is injured you may develop DI.

**S/s:** polydipsia, polyuria. Nocturia, specific gravity of 1.001-1.005.

Fluid volume deficit: weight loss, poor skin turgor, dry mucous membranes, hypotension, tachycardia, weak/poor peripheral pulses, decreased cognition, ataxia, dry cracked lips.

**Labs**: 24-hour urine collection (volume and creatinine), vasopressin challenge, and fluid deprivation test. You will have dilute urine and concentrated blood.

**Evaluation of treatment success for diabetes insipidus**

-you know it’s working when they stop peeing excessively, Na+ decreases and pressure improves.

**SIADH causes, labs, and management**

-excessive secretion of ADH, causing significant water retention

**Causes:** pulmonary disorders, malignancies, disorders of the nervous system, and meds (chemotherapy agents, TCA’s, SSRI’s, opiods, fluoroquinolone antibiotics), head injury, T, meningitis, stroke

**Labs: concentrate urine with dilute blood.** hyponatremia (less than 134), low bun (less than 10), hypouricemia (below 4)

**S/s:** normal vitals, moist mucous membranes, normal skin turgor, no edema (the water has moved from the extra cellular space to the intracellular space), confusion, lethargy, weakness, asterixis, seizure, coma

**Management:** fluid restriction (less than 1 L per day), 3% NaCl (hypertonic solution) if Na+ is less than 120, furosemide but only if Na+ is at least 125, monitor Na+, signs of hyponatremia such as confusion, seizures, and delirium, cardiopulmonary status – HTN, crackles, tachypnea, decreased O2 sats, ascites, JVD, peripheral edema, head of the bed should not be raised more than 10 degrees.

**S/s of hypocalcemia**

-tetany: tingling of fingers and toes

-carpopedal spasms

-convulsions

-positive Chvostek’s (face tap) and Trousseau’s (BP cuff) sign.

**Medical treatment of hyperparathyroidism**

-removal of abnormal tissue: parathyroidectomy

-2000 mL daily fluid intake helps prevent kidney stones

-avoid thiazide diuretics

-report abdominal pain, hematuria, vomiting and diarrhea

**Pancreatitis s/s, common causes, complications, nursing interventions, medical treatment, diet**

S/s: sudden onset of severe pain epigastric, LUQ radiating around body, bruising on flank and around navel, jaundice, decreased or absent bowel sounds, ascites, tetany due to hypocalcemia

Common Causes: biliary tract disease, alcoholism, hypertriglyceridemia, trauma, GI surgery

Diet: NPO. When resumed bland, high protein, low fat, with no stimulants such as caffeine.

Nursing Interventions:

-no alcohol, smoking or stress

-pain meds

-provide insulin as needed

-administer fluid and electrolytes

-monitor hydration status

-position in comfortable position fetal, leaning forward

Medical Treatment

-morphine, hydromorphone, ketorolac

-antibiotics: imipenem

-ranitidine: decreases gastric acid

-PPI: omeprazole – decreases gastric acid secretion

-pancrelipase: aids with digestion of fat and proteins when taken with meals and snacks

Complications

-hypovolemia

-pancreatic infection

-DM1

-atelectasis: more common in older adults

-DIC

-Multi system organ failure

**Liver failure treatment, s/s, paracentesis procedure, complications, treatment of esophageal varices bleeding, management of ascites, hepatic encephalopathy treatment and diet**

Liver failure treatment

-diuretics: get rid of excess fluid

-beta blockers: to prevent bleeding from varices

-lactulose: excrete excess ammonia

-paracentesis: treats the ascites

-liver transplant

Liver Failure S/s

-Abdominal pain

-dyspepsia

-constipation or diarrhea

-weight loss, ab pain, and distention

-ascites

-varices

-splenomegaly from back up of blood

-decreased plasma albumin

-increased aldosterone

-clotting problems

-anemia, jaundice, pruritus

-neurologic changes: confusion from buildup of waste

-musty breath

Paracentesis procedure: removal of fluid from the peritoneal cavity

-used for diagnostic reasons

-treatment of massive ascites

-have PT void before procedure

-observe for vascular collapse

Paracentesis complications

-ascitic fluid leak

-hemorrhage

-infection

-perforation

esophageal varices treatment:

**Cause**: portal hypertension

**Treatment**:

-beta blocker (Inderal – decrease HR and pressure in portal circulation) – good for preventing the bleeding not for an acute bleed

-vasopressin (watch out for constriction of coronary vessels)

-esophagogastric balloon

-TIPS (shunt from portal vein to hepatic vein which bypasses the liver)

-somatostatin: octreotide like vasopressin but without vasoconstriction

-fluid, electrolytes, and blood transfusion

Management of Ascites

-diuretics: spironolactone. Add furosemide if ascites doesn’t resolve.

-low salt diet

-bed rest

-paracentesis

Hepatic encephalopathy Treatment and Diet

**S/s**: mental status change and motor disturbances. Not oriented. Coma. Seizures.

**Treatment**: lactulose to get rid of excess ammonia, IV glucose to minimize protein breakdown.

-antibiotics

-fluid and electrolytes

**Diet**:

-Protein 1.2 – 1.5 g/kg/day

-Enteral feeding

**Hepatitis transmission and prevention, diet for Hepatitis A**

Transmission

A – fecal/oral

B – blood

C – blood

D – coinfection with HBV

E – oral/fecal

Prevention

-vaccination

-wash hands before preparing or eating food

-safe injection practices

-drink purified, don’t share utensils or bed linens in underdeveloped countries

Diet for Hep A

-wash fruits and veggies

-cook shellfish

-drink purified water in underdeveloped countries

**Cholelithiasis (gall stones) treatment, s/s, diet, laboratory**

S/s: indigestion, moderate to severe pain, fever, jaundice, RUQ pain radiating to right shoulder, n/v, restlessness, diaphoresis, dark amber urine, jaundice, clay-colored stools, tachypnea, Murphy’s sign, Blumberg’s sign, steatorrhea (fatty stools)

Labs:

-liver function studies AST and ALT increased with common bile duct obstruction

-WBC count elevated - inflammation

-Serum bilirubin – increased when bile duct is obstructed

-serum amylase – increased when pancreas involved

Treatment

-bile acids: dissolve cholesterol-based stones

-ERCP: common in removing common bile duct stones

-ESWL: used if stone is too large to pass

-cholecystectomy

-pain meds: morphine or hydromorphone and ketorolac

-fat soluble vitamins ADEK

Diet

-reduced calories if obese

-low saturated fat

-high fiber and Ca+

-take fat soluble vitamins

-avoid gas forming foods such as cabbage, beans, cauliflower, broccoli.