NR507 Advanced Pathophysiology FINAL EXAM MASTER STUDY GUIDE

Bullet-by-bullet high-yield review with pathophysiology, symptoms, diagnostics, treatments, mnemonics, comparison tables, and exam pearls.

How to use this: memorize the comparison tables and mnemonics first, then read the pathophysiology paragraphs to understand why the symptoms, labs, and treatments happen. Multiple choice questions usually test mechanisms and differentiation, not memorized definitions.

FINAL EXAM QUICK MAPS

GI: inflammation, obstruction, bleeding, and liver failure
GERD = weak LES -> acid exposure -> esophagitis
Appendicitis = lumen obstruction -> bacterial overgrowth -> ischemia/perforation risk
PUD = acid/pepsin injury > mucosal defense, usually H. pylori or NSAIDs
IBD = inappropriate immune inflammation of bowel; UC mucosa/colon, Crohn transmural/skip lesions
Cirrhosis = fibrosis -> portal HTN + low albumin -> ascites and encephalopathy

Endocrine: read the feedback loop
Primary gland failure = pituitary hormone rises to compensate, example high TSH in hypothyroidism
Overactive target gland = pituitary hormone suppressed, example low TSH in hyperthyroidism
Cortisol excess = catabolism + glucose elevation + immune suppression
Parathyroid hormone raises calcium and lowers phosphate

Neuro: location explains symptoms
CNS demyelination = MS, scattered CNS deficits
Peripheral demyelination = GBS, ascending weakness/areflexia
NMJ transmission problem = myasthenia gravis, fatigable weakness
Cerebral blood flow interruption = TIA/stroke symptoms

WEEK 5 - GASTROINTESTINAL PATHOLOGIES

Pathophysiology of GERD

Pathophysiology: GERD occurs when the lower esophageal sphincter is weak or relaxes inappropriately. Gastric acid and pepsin reflux upward into the esophagus. The esophagus does not have the same protective mucus and bicarbonate barrier as the stomach, so repeated acid exposure causes irritation, inflammation, erosions, and possible metaplasia over time.

Signs and symptoms:

• Burning substernal heartburn after meals or when lying down

• Sour/bitter regurgitation

• Chronic cough, hoarseness, throat clearing, globus sensation, wheezing from microaspiration or vagal stimulation

• Dysphagia or odynophagia are alarm features and need evaluation

Diagnostics/labs:

• Usually clinical when classic symptoms are present

• Endoscopy if alarm symptoms, bleeding, weight loss, anemia, dysphagia, persistent vomiting, or refractory symptoms

• pH monitoring confirms abnormal acid exposure when diagnosis is unclear

Treatment/management:

• Lifestyle: weight loss when indicated, elevate head of bed, avoid late meals, reduce trigger foods, stop smoking

• Pharmacologic: proton pump inhibitors are strongest acid suppression; H2 blockers can help mild/intermittent symptoms; antacids give quick short-term relief

Memory clue: GERD = Gate (LES) opens -> Esophagus gets acid -> Reflux disease.

Exam pearl: GERD is not too much acid only. The key issue is acid in the wrong place because the LES barrier fails.

Risk factors for esophageal stricture

Pathophysiology: An esophageal stricture forms when chronic injury heals with fibrosis and scarring. In GERD, repeated acid damage causes inflammation, ulceration, collagen deposition, and narrowing of the lumen.

Signs and symptoms:

• Progressive solid food dysphagia first, then liquids if severe

• Food sticking sensation

• History of chronic GERD or caustic/radiation injury

Diagnostics/labs:

• Endoscopy evaluates narrowing and rules out malignancy

• Barium swallow may show narrowed segment

Treatment/management:

• Acid suppression with PPI

• Endoscopic dilation when symptomatic

• Evaluate alarm features because cancer can also cause progressive dysphagia

Exam pearl: Progressive dysphagia is never a throwaway symptom. It means obstruction until proven otherwise.

Hiatal hernia treatment

Pathophysiology: A hiatal hernia occurs when part of the stomach moves upward through the diaphragm. This disrupts the gastroesophageal junction and makes reflux more likely.

Signs and symptoms:

• Heartburn, regurgitation, chest discomfort, dysphagia

• Many sliding hernias are asymptomatic

Diagnostics/labs:

• Endoscopy, barium swallow, or imaging may identify hernia if symptoms warrant

Treatment/management:

• Lifestyle measures and acid suppression for reflux symptoms

• Surgical repair/fundoplication for large paraesophageal hernia, obstruction, strangulation risk, bleeding, or refractory GERD

Exam pearl: Sliding hiatal hernia commonly causes GERD. Paraesophageal hernia is more concerning for strangulation/obstruction.

Pathophysiology of appendicitis

Pathophysiology: Appendicitis usually begins with obstruction of the appendiceal lumen by fecalith, lymphoid hyperplasia, tumor, or foreign body. Mucus continues to be secreted into a closed space, intraluminal pressure rises, venous outflow is impaired, bacteria multiply, ischemia develops, and the appendix can perforate.

Signs and symptoms:

• Early vague periumbilical pain from visceral nerve irritation

• Pain migrates to right lower quadrant as parietal peritoneum becomes inflamed

• Anorexia, nausea/vomiting, low-grade fever

• McBurney point tenderness, rebound, guarding, positive Rovsing/psoas/obturator signs may occur

Diagnostics/labs:

• CBC: leukocytosis with neutrophilia

• CRP may be elevated

• Urinalysis helps rule out urinary causes but mild pyuria can occur from irritation

• Pregnancy test in reproductive age patients

• CT abdomen/pelvis commonly used in adults; ultrasound often used first in children/pregnancy

Treatment/management:

• Appendectomy is definitive for many patients

• Antibiotics and IV fluids; nonoperative antibiotics may be considered in selected uncomplicated cases

• Perforation/abscess may require drainage plus antibiotics before or with surgery

Exam pearl: Periumbilical pain that migrates to RLQ is classic because visceral pain becomes localized parietal peritoneal pain.

Risks for appendectomy in adults

Pathophysiology: Risk comes from the condition and the surgery. Delayed appendicitis increases perforation and abscess risk. Adult surgical risk is higher with comorbidities and complicated disease.

Signs and symptoms:

• Older age, obesity, diabetes, immunosuppression, pregnancy, anticoagulation, cardiovascular/pulmonary disease

• Perforated appendicitis, abscess, sepsis, delayed presentation

Diagnostics/labs:

• Pre-op labs, imaging, pregnancy testing when relevant, anesthesia assessment

Treatment/management:

• Early recognition and antibiotics decrease complications

• Post-op monitoring for wound infection, abscess, ileus, bleeding, and sepsis

Exam pearl: Adults may present atypically. Do not rely on a perfect textbook presentation.

Peptic ulcer disease pathophysiology

Pathophysiology: PUD occurs when gastric acid and pepsin overwhelm mucosal defenses. H. pylori damages mucosa and promotes inflammation. NSAIDs inhibit prostaglandins, decreasing mucus, bicarbonate, mucosal blood flow, and repair. The result is erosion that extends through the muscularis mucosae.

Signs and symptoms:

• Epigastric burning/gnawing pain

• Nausea, bloating, early satiety

• Melena or hematemesis if bleeding

• Sudden severe abdominal pain if perforation

Diagnostics/labs:

• H. pylori testing: urea breath test, stool antigen, biopsy during endoscopy

• CBC if bleeding/anemia suspected

• Endoscopy for alarm symptoms or older age/new onset symptoms

Treatment/management:

• Stop NSAIDs if possible

• PPI therapy

• H. pylori eradication with combination antibiotics plus acid suppression

• Avoid smoking and excessive alcohol

Exam pearl: H. pylori and NSAIDs are the big two. NSAIDs remove the stomach lining protection; H. pylori inflames and injures the lining.

Compare gastric and duodenal ulcers

Feature	Gastric ulcer	Duodenal ulcer
Pain timing	Often worse with meals because food stimulates acid against injured stomach mucosa	Often improves with meals then returns 2-3 hours later or at night
Weight pattern	May cause weight loss due to food avoidance	May cause weight gain or night eating due to pain relief with food
Acid pattern	Normal or low acid can occur; mucosal defense failure is key	Often increased acid load to duodenum
Cancer concern	Biopsy often important because gastric ulcers can be malignant	Malignancy is less common
Classic clue	Epigastric pain shortly after eating	Pain wakes patient at night and improves after food/antacid

Memory clue: Gastric = Grumbles with grub. Duodenal = Decreases with dinner but returns later.

Causes of gastric ulcers

Pathophysiology: Gastric ulcers happen when protective mucosal barriers are damaged. NSAIDs reduce prostaglandin-mediated mucus/bicarbonate protection. H. pylori creates chronic gastritis and mucosal injury. Severe physiologic stress, bile reflux, smoking, and malignancy can contribute.

Signs and symptoms:

• Epigastric pain worse with eating, nausea, early satiety

• Occult bleeding or melena

Diagnostics/labs:

• Endoscopy with biopsy when indicated

• H. pylori testing

Treatment/management:

• Remove cause, PPI, eradicate H. pylori, avoid NSAIDs

Exam pearl: Gastric ulcers are not always from high acid; they are often from weak protection.

Signs and symptoms of duodenal ulcer

Pathophysiology: Duodenal ulcers form in the first part of the duodenum, usually from H. pylori-related increased acid effect or impaired bicarbonate defense.

Signs and symptoms:

• Burning epigastric pain 2-3 hours after meals

• Nocturnal pain

• Pain improves temporarily with food or antacids

• Melena or anemia if bleeding

Diagnostics/labs:

• H. pylori testing

• Endoscopy if alarm symptoms or refractory disease

Treatment/management:

• PPI plus H. pylori eradication when positive

• Avoid NSAIDs

Exam pearl: Pain better with meals is a high-yield duodenal ulcer clue.

Pathophysiology of ulcerative colitis

Pathophysiology: Ulcerative colitis is chronic immune-mediated inflammation limited to the colon and rectum. It begins in the rectum and extends continuously. Inflammation is usually mucosal and submucosal, causing friability, superficial ulcers, bleeding, and loss of absorptive function.

Signs and symptoms:

• Bloody diarrhea, urgency, tenesmus

• LLQ cramping, fever during flares, fatigue

• Extraintestinal manifestations: arthritis, uveitis, skin lesions, primary sclerosing cholangitis

Diagnostics/labs:

• Colonoscopy with biopsy shows continuous inflammation

• Stool studies rule out infection during flares

• CBC may show anemia/leukocytosis; CRP/ESR may be elevated

Treatment/management:

• 5-ASA for mild-moderate disease depending on extent

• Corticosteroids for flares, not long-term maintenance

• Immunomodulators/biologics for moderate-severe disease

• Colectomy can be curative for colonic disease

Exam pearl: UC = Uniform and Continuous in Colon.

Risk factors for ulcerative colitis and Crohn disease

Pathophysiology: IBD results from genetic susceptibility, epithelial barrier dysfunction, abnormal immune response to gut microbiota, and environmental triggers.

Signs and symptoms:

• Family history of IBD

• Ashkenazi Jewish ancestry has increased risk

• Smoking increases Crohn risk but is complex in UC

• Urban/industrialized environment, prior GI infections, dysbiosis

Diagnostics/labs:

• Diagnosis uses history, colonoscopy/biopsy, inflammatory markers, stool testing, and imaging for Crohn complications

Treatment/management:

• Treatment is based on disease severity, location, complications, and flare vs maintenance

Exam pearl: IBD is immune dysregulation, not simply infection.

Symptoms of ulcerative colitis

Pathophysiology: Mucosal inflammation makes the colon unable to absorb water and causes bleeding from friable ulcerated mucosa.

Signs and symptoms:

• Bloody diarrhea

• Urgency and tenesmus from rectal inflammation

• Crampy abdominal pain, often LLQ

• Fatigue, weight loss, fever in severe flares

• Severe complication: toxic megacolon

Exam pearl: Blood and tenesmus point strongly toward rectal/colonic mucosal inflammation.

Pathophysiology of Crohn disease

Pathophysiology: Crohn disease is chronic immune-mediated inflammation that can affect any part of the GI tract from mouth to anus. It is transmural, meaning inflammation goes through the bowel wall. This causes deep ulcers, strictures, fistulas, abscesses, and skip lesions.

Signs and symptoms:

• Chronic diarrhea, often nonbloody

• RLQ pain if terminal ileum involved

• Weight loss, fever, fatigue

• Perianal disease, fistulas, abscesses

• Malabsorption, especially B12 and bile salts if ileum involved

Diagnostics/labs:

• Colonoscopy with biopsy may show skip lesions/cobblestoning

• CT/MR enterography evaluates small bowel and fistulas

• CBC, CRP/ESR, stool studies

Treatment/management:

• Steroids for acute flares

• Immunomodulators/biologics for maintenance or moderate-severe disease

• Antibiotics for abscess/perianal complications when indicated

• Surgery treats complications but is not curative

Exam pearl: Crohn = Can occur anywhere, Cobblestone, Creeps through entire wall.

Crohn disease vs ulcerative colitis

Feature	Crohn disease	Ulcerative colitis
Location	Mouth to anus; terminal ileum common	Colon and rectum only
Pattern	Skip lesions	Continuous from rectum proximally
Depth	Transmural	Mucosal/submucosal
Stool	Often nonbloody diarrhea	Bloody diarrhea common
Complications	Fistulas, strictures, abscesses, malabsorption	Toxic megacolon, colorectal cancer risk
Surgery	Not curative; disease can recur	Colectomy can be curative for colitis

Pathophysiology of non-alcoholic fatty liver disease

Pathophysiology: NAFLD is hepatic fat accumulation not caused by significant alcohol use. Insulin resistance increases free fatty acid delivery to the liver and promotes triglyceride accumulation in hepatocytes. Oxidative stress and inflammation can progress to nonalcoholic steatohepatitis, fibrosis, cirrhosis, and liver failure.

Signs and symptoms:

• Often asymptomatic

• Fatigue, RUQ discomfort, hepatomegaly may occur

Diagnostics/labs:

• Mild ALT/AST elevation; imaging shows steatosis

• Evaluate metabolic syndrome and exclude other causes

Treatment/management:

• Weight loss, exercise, control diabetes/lipids, avoid alcohol and hepatotoxins

Exam pearl: NAFLD is the liver manifestation of metabolic syndrome.

Pathophysiology of alcoholic cirrhosis

Pathophysiology: Chronic alcohol use causes acetaldehyde toxicity, oxidative stress, inflammation, fatty change, hepatocyte injury, and stellate cell activation. Stellate cells lay down collagen, forming fibrosis and regenerative nodules. This distorts blood flow and decreases liver function.

Signs and symptoms:

• Fatigue, anorexia, weight loss, jaundice

• Spider angiomas, palmar erythema, gynecomastia, testicular atrophy

• Ascites, varices, splenomegaly, encephalopathy