GASTROINTESTINAL AND HEPATOBILIARY SYSTEMS

The focus of this week’s lab will be pathology of the gastrointestinal and hepatobiliary systems.

GASTROINTESTINAL SYSTEM AND HEPATOBILIARY SYSTEM

We will examine the pathology of the gastrointestinal tract and the hepatobiliary system. This section includes many different organs. Make sure that you know them and their basic functions and it will help you understand what is occurring at the cellular level.

In general, the wall of the intestinal tract is comprised of three layers: 1) the mucosa, 2) the submucosa, and 3) the muscularis externa. Changes to these layers due to pressure, inflammation, or erosion can result in pathologic conditions.

The cases we will cover are:

A. Peptic Ulcer Disease
B. Crohn’s Disease
C. Diverticulosis
D. Cirrhosis
E. Zollinger-Ellison Syndrome

A. PEPTIC ULCER DISEASE

CC/HPI: A 25 year old man complains of epigastric pain that usually begins one to two hours after eating and occasionally awakens him at night. The patient has been diagnosed with duodenal ulcers several times in the past, but his symptoms have consistently recurred even after therapy with histamine blockers, antacids, and sucralfate.

PE: Vital signs normal. Physical exam reveals pallor; epigastric tenderness on deep palpation.

Labs: CBC demonstrates normochromic, normocytic anemia. Stool positive for occult blood. UGI: ulcerations of antrum of stomach and duodenum; urea breath test is positive.

Pathology: Grossly round ulcer seen as sharply punched-out defect with relatively straight walls and slight overhanging of mucosal margin (heaped-up margin characteristic of malignant lesion); smooth and clean ulcer base. No evidence of malignancy; antral biopsies reveal presence of chronic mucosal inflammation and necrotic tissue; organisms identified on methylene blue stain.

What are the three major layers of the gut wall?

Which layers are affected in this patient?
What organism is seen in the methylene blue stained image?

What enzyme is needed for this organism to survive in the acidic pH of the stomach?

What is “triple therapy” that is used to treat this infection?

B. CROHN’S DISEASE

CC/HPI: A 21 year old woman complains of intermittent abdominal pain, mild, non-bloody diarrhea, and anorexia for the past two years. She states that the pain is almost always confined to the right lower abdomen and is cramping in nature.

PE: Physical exam reveals pallor; weight loss; abdominal mass in right iliac fossa (thickened bowel loop).

Labs/Imaging: CBC demonstrates megaloblastic anemia. Guaiac positive stool, but negative for parasites. BE: granulomatous colitis and regional enteritis involving multiple areas, most commonly ileum and ascending colon.

Why does this patient have megaloblastic anemia? (Hint! What vitamin deficiencies result in megaloblastic anemia? Can you guess which vitamin she is deficient in based on her symptoms?)
What area of the gastrointestinal tract is likely involved in this anemia? (Hint! Where is this vitamin absorbed?)

Pathology: Terminal ileum shows lesions that have a “cobblestone” appearance; discontinuous areas of inflammation, edema, and fibrosis (“skip lesions”). Chronic inflammatory involvement of mucosal, submucosal, and muscularis layers of bowel wall (transmural inflammation), manifested mainly by lymphocytic infiltration with associated lymphoid hyperplasia and formation of non-caseating granulomas.

What are the three major layers of the gut wall?

What layers of the intestinal wall are inflamed in this patient?

C. DIVERTICULOSIS

CC/HPI: A 55 year old woman presents with lower abdominal discomfort, chronic constipation, and occasional passage of bright red blood per rectum. She is a heavy smoker, and her diet contains a significant amount of greasy food and little fiber.

What diets contribute to diverticulosis?

PE: Vital signs normal. Physical exam reveals pallor, mild left abdominal tenderness with palpable descending colon; guaiac positive stool on rectal exam.
Labs/Imaging: CBC reveals normochromic, normocytic anemia. No blood in stool, no leukocytes or epithelial cells seen. Sigmoidoscopy reveals multiple small outpouchings in the walls of descending and sigmoid colon without inflammation.

Pathology: Multiple flasklike outpouchings less than a centimeter in size along taeniae coli in walls of descending and sigmoid colon. Thin-walled herniations of atrophic mucosa and compressed submucosa; hypertrophied circular layer of muscularis propria with prominent taeniae coli.

What gut layers are present in the normal colonic wall?

What two layers of the gut are present in this diverticuli?

What complications are associated with diverticulosis?

D. CIRRHOSIS

CC/HPI: A 50 year old man with a history of alcoholism is unresponsive to stimuli when brought by a neighbor to the emergency room. His neighbor states that he had vomited blood (hematemesis) three months ago but had received no treatment. The neighbor also reports that the patient got drunk three times a week for four years until approximately one year ago.

PE: Muscle wasting; icteric sclera; spider angiomata (due to increased levels of estrogen); nodular, hard hepatomegaly; caput medusa; loss of hair on chest and genitalia; ascites; gynecomastia; testicular atrophy; parotid enlargement; flapping tremor of hands (asterixis); palmar erythema; slight pitting edema in lower extremities.

Why are spider angiomata present in this patient?

Why is caput medusa present in this patient?
Labs/Imaging: CBC/CMP: slight thrombocytopenia; macrocytic anemia. Increased bilirubin; elevated serum transaminase and alkaline phosphatase; low serum albumin with increased globulins; prolonged PT; high blood ammonia. UGI: esophageal varices. EGD: esophageal varices confirmed. CT/US, abdomen: enlarged and fatty nodular liver; tortuous, dilated variceal vessels.

Pathology: Early: enlargement and fatty infiltration of liver; late: brownish discoloration, hardening, and atrophy of liver parenchyma. Necrosis of normal hepatocytes; diffuse replacement with fibrous connective tissue and lymphocyte infiltrate; regenerating nodules of liver lacking normal organization; eosinophilic Mallory bodies; bile congested ductules and proliferation of fibroblasts.

What tissue is this?

What are the pink inclusions in the cells in the right picture called?

What is the nodule seen in the picture on the left?

E. ZOLLINGER-ELLISON SYNDROME

CC/HPI: A 43 year old white man complains of severe burning epigastric pain and diarrhea of two years duration that has been refractory to medical management. The pain awakens him early in the morning, is accompanied by nausea and vomiting, increases with coffee consumption, and also appears two to three hours after meals. Three days ago he also noticed black stools.

PE: Slight discomfort on epigastric palpation but no signs of peritoneal irritation; pale skin and mucous membranes; occult blood on digital rectal examination.

Labs/Imaging: Fasting serum gastrin markedly increased; increased gastric acid output (hyperchlorhydria).
What cells usually secrete gastrin? What is the stimulus for gastrin secretion?

What is the function of gastrin?

Pathology: Gross pathology indicates ulcers in uncommon places in esophagus, duodenum, and jejunum; gastrinoma in pancreas. Usually originates from delta cells of pancreas; original lesion may be an adenoma, hyperplasia, or carcinoma. Immunocytochemistry indicates gastrin present in adenoma.

How does unregulated gastin release by the pancreas result in ulcers in this patient?

What tissue is this?

What is abnormal about its histological appearance? Note the right side of the image shows normal tissue and the left is abnormal.