DISORDERS OF GASTROINTESTINAL FUNCTIONAL

CHAPTER 39
OTHER PEOPLE THINK ABOUT ME....

....THEREFORE I AM.
IRRITABLE BOWEL SYNDROME

“A functional GI disorder characterized by a variable combination of chronic and recurrent intestinal symptoms not explained by structural or biochemical abnormalities.”

SYMPTOMS:

- Abdominal pain, altered bowel function, flatulence, bloating, anorexia, nausea, constipation, diarrhea.
IRRITABLE BOWEL SYNDROME

- Sx’s relieved by defecation.
- Associated with a change in consistency or frequency of stools.
- PAIN: cramping, intermittent, lower abdominal. **Does not occur at night or interfere w/ sleep.**
- Cause: Not clear. Felt to be a “dysregulation” of intestinal motor and sensory functions modulated by the CNS.
- **STRONG** psychogenic component.
IRRITABLE BOWEL SYNDROME

- **DIAGNOSIS**: A diagnosis of exclusion.
- Consider “organic” disease with:
  - Acute onset, weight loss, anemia, occult blood, **symptoms at night**, evidence of malabsorption.
INFLAMMATORY BOWEL DISEASE

- CROHN’S DISEASE. (CROHN DISEASE)
- ULCERATIVE COLITIS.
INFLAMMATORY BOWEL DISEASE

- Distinct disorders, but share:
  - 1) Inflammation of the bowel.
  - 2) Lack a proven causative agent.
  - 3) Have a pattern of familial occurrence.
- Results from: activation of inflammatory cells and elaboration of inflammatory mediators, causing non-specific tissue damage.
INFLAMMATORY BOWEL DISEASE

CHARACTERIZED BY

- Remissions and exacerbations of diarrhea, fecal urgency, weight loss.
- Systemic manifestations—see text—arthritis, skin lesions, anemia, etc.
INFLAMMATORY BOWEL DISEASE

**CAUSE**

- “Largely unknown.”
- Best guess: a genetic susceptibility that triggers some form of autoimmune reaction. Triggered by a dietary antigen or microorganism???
CROHN’S DISEASE

- Recurrent, granulomatous inflammation.
- “Tongue to bung.”
- 30% - small bowel only.
- 30% - large bowel only.
- 30% - both.
- Sharply demarcated lesions, “skip” lesions.
- All layers of the bowel wall affected, especially the submucosa.
- Fibrosis, thickening.
CROHN’S DISEASE

- Effects females slightly more than males.
- 20’s, 30’s. Onset in some in adolescence.
CROHN’S DISEASE

CLINICAL COURSE

- Exacerbations and remissions.
- Intermittent diarrhea, cramping pain, weight loss, fluid & electrolyte disorders, low-grade fever.
- Bloody diarrhea, but less than in ulcerative colitis.
CROHN’S DISEASE

**COMPLICATIONS**

- Malabsorption, nutritional deficiencies.
- Fistula formation.
- Abscess formation.
- Intestinal obstruction.
CROHN’S DISEASE

DIAGNOSIS

- Sigmoidoscopy.
- Upper GI w/ small bowel follow through (UGI W/ SBFT), barium enema (BE) – done to evaluate extent of disease, presence of fistulae.
- CT- to detect abscess formation.
ULCERATIVE COLITIS.

- Non-specific inflammation.
- Confined to the colon and rectum.
- Begins in the rectum, spreads proximally.
- Mucosa primarily involved.
- Continuous rather than patchy (no skip lesions).
ULCERATIVE COLITIS.

**CLINICAL COURSE**

- Remissions and exacerbations.
- Diarrhea, often bloody, w/ mucous.
- Mild abdominal cramping, fecal incontinence, anorexia, weakness, fatigue.
- Course can be mild to fulminant.

**COMPLICATIONS**

- Increased risk of colon cancer.
ULCERATIVE COLITIS.

DIAGNOSIS

- Proctosigmoidoscopy
DIVERTICULAR DISEASE

- DIVERTICULOSIS.
- DIVERTICULITIS.

**RISK FACTORS FOR:**
- 1) Dietary- lack of fiber.
- 2) Decreased physical activity.
- 3) Poor bowel habits.
- 4) Aging.
APPENDICITIS

- Common.
- Most frequently seen in the 5-30 year-old age group.
- Delayed diagnosis in the elderly, increased morbidity and mortality.
- Due to obstruction of the appendix, usually by a fecalith.
- Perforates if not treated.
APPENDICITIS

PRESENTATION

- "Uncommon presentations of common disorders are more common than common presentations of uncommon disorders."
- Abrupt onset, brief.
- Periumbilical or epigastric pain that shifts to the RLQ.
- Nausea, anorexia, vomiting, diarrhea.
- Leukocytosis, neutrophilia (neutrophils, PMN’s, poly’s).
- Tenderness to palpation in the RLQ (McBurney’s Point) w/ or w/out rebound tenderness.
DIARRHEA

- CAUSES
- 1) INFECTION.
- 2) FOOD INTOLERANCE.
- 3) DRUGS.
- 4) INTESTINAL DISEASES: inflammatory bowel disease, irritable bowel syndrome, malabsorption syndrome, endocrine disorders (hyperthyroidism, diabetic neuropathy), radiation colitis.
DIARRHEA

**ACUTE**
- Lasts less than 4 days, Self-limiting.
- Usually infectious.

**CHRONIC**
- Longer than 3-4 weeks.
- Usually caused by intestinal disorders.
DIARRHEA

- LARGE-VOLUME DIARRHEA.
  - Results from an increase in water content of the stool. 2 TYPES:
  - 1) SECRETORY.
  - 2) OSMOTIC.

- SMALL-VOLUME DIARRHEA.
  - Results from an increase in peristalsis.
  - See chart 39-1, Pg 904.
FECAL IMPACTION

- Most common in the debilitated elderly.
- Causes:
  - Painful anorectal diseases, tumors, neurogenic disease, drugs, low-residue diet, prolonged bed rest, combination.
- Symptoms: abdominal pain, watery diarrhea, fecal incontinence, abdominal distention, urinary incontinence.
MALABSORPTION SYNDROME

3 TYPES OF MALABSORPTION

1) INTRALUMINAL MALDIGESTION.
2) TRANSEPIPTHELIAL TRANSPORT.
3) LYMPHATIC OBSTRUCTION.

MOST COMMON CAUSES

1) PANCREATIC INSUFFICIENCY.
2) HEPATOBILIARY DISEASE.
3) INTRALUMINAL BACTERIAL GROWTH.
MALABSORPTION SYNDROME

- SYMPTOMS: steatorrhea, diarrhea, cramping, distention, flatulence, bloating.
- ALSO: weight loss, muscle wasting, weakness.
- STEATORRHEA: stools from the malabsorption of fat. Yellowish, malodorous, bulky, float don’t flush.
- ALSO MALABSORPTION OF FAT-SOLUBLE VITAMINS: K, D.
CELIAC DISEASE

- See text, Pg 908.
COLORECTAL CANCER

- 2ND LEADING CAUSE OF CANCER DEATH IN THE U.S.

**RISK FACTORS**

- 1) AGE- over 50.
- 2) GENETIC.
- 3) OTHER DISORDERS- inflammatory bowel disease, familial polyposis.
- 4) DIET- high fat & sugar, low fiber, deficient in Vitamins A, C, E.
COLORECTAL CANCER

SYMPTOMS

- Malignancy usually present a long time before Sx’s develop.
- Painless hematochezia.
- Change in bowel habits, diarrhea, constipation, decreased stool caliber (late).
COLORECTAL CANCER

EARLY DETECTION / SCREENING

- The ticket to lowering mortality
- Colonoscopy / sigmoidoscopy - the gold standard
- Hemoccult, “Guaiac” test - at time of annual exam, home kits.
- Barium enema.
COLORECTAL CANCER

CHEMOPREVENTION

- To be covered in detail in Biomedical Rx of Disease I.
- Calcium, aspirin, NSAID’s.
CHAPTER 40
The patient's bill of rights gets watered down.

You have the right not to say "ah".
THE VIRAL HEPATITIDES

- HEPATITIS A, B, C.
- ALSO D, E, & G.
- DIFFER RE: mode of transmission, incubation period, degree of liver damage, chronic / carrier state.

- 2 MECHANISMS OF HEPATOCellular INJURY:
  - 1) Direct cellular injury.
  - 2) Immune Response against the virus.
THE VIRAL HEPATITIDES

- The higher the degree of the immune response, the more cellular damage, but the higher the degree of eradication of the virus, and the less likely the chance of chronic infection and carrier state.

**THE INFECTION:**
- 1) The Prodromal / pre-icteric phase.
- 2) The icteric phase.
- 3) The convalescent phase.
HEPATITIS A

- TRANSMISSION: fecal-oral route. Not by blood or sex.
- INCUBATION PERIOD: brief, 2-6 weeks.
- CLINICAL COURSE: abrupt onset, fever, malaise, nausea, anorexia, abdominal pain, jaundice (often profound), dark urine.
- NO CHRONIC / CARRIER STATE.
- VACCINATION: available.
HEPATITIS B

- TRANSMISSION: blood and body fluids; needles, sexual and oral contact; perinatal.
- INCUBATION PERIOD: longer than Hep A.
- CLINICAL COURSE:
  1) Acute hepatitis.
  2) Chronic hepatitis → cirrhosis.
  3) Fulminant hepatitis, hepatic necrosis.
  4) Carrier state.
HEPATITIS B

- SEROLOGIC MARKERS:
  1) HBsAg- hepatitis B surface antigen.
  2) HBeAg- the e antigen.
  3) HBcAg- the core antigen.
HEPATITIS B

VACCINE

- Available. Now recommended for all children and for those at risk: healthcare workers; clients and staff of institutions for the disabled; recipients of blood products; dialysis patients; those from endemic countries; IV users; inmates; sex: men with men, all those w/multiple partners.

- PREGNANCY- routine screening for HBsAg.
Many / most patients with hepatitis B are chronic carriers with no symptoms, whose disease is found during an evaluation for elevated liver enzymes. Those who become infected but do not become chronic carriers often have a clinical course that is very mild or even completely asymptomatic.
HEPATITIS C

- Most common cause worldwide of chronic hepatitis, cirrhosis, and hepatocellular carcinoma.
- Previously known as Non-A Non-B up until about 1991.
- Most are chronically infected and unaware, not ill.
- 6 genotypes- severity depends on genotype. Multiple genotypes accounts for elusive nature from the immune system and the inability to develop a vaccine.
HEPATITIS C

- TRANSMISSION - primarily via needles. Sexual and perinatal transmission possible, but "incidence is uncertain."
- INCUBATION PERIOD: averages 50 days.
- CLINICAL COURSE - usually asymptomatic, or mild illness w/ nonspecific Sx’s. Jaundice is uncommon. Fulminant hepatitis is rare. Chronic hepatitis and cirrhosis, carcinoma.
- NO VACCINE.
ALCOHOL-INDUCED LIVER DISEASE

- 3 EXCITING FLAVORS:
  - 1) Fatty liver disease.
  - 2) Alcoholic hepatitis.
  - 3) Cirrhosis.
- See text re metabolism of alcohol, Cytochrome P450, acetaldehyde, etc. Pg 933.
- See text re the histopathologic changes in the liver, Pg 933-934 (fibrosis, scarring).
CIRRHOSIS

- Replacement of functional liver tissue by fibrosis.
- Results in obstruction of vascular and lymphatic channels.
- This results in:
  1) Portal hypertension.
  2) Biliary obstruction, exposing the liver cells to destructive effects of bile stasis.
  3) Loss of Liver cells.
CIRRHOSIS

- Usually due to alcohol.
- But can be due to: viral hepatitis, hepatotoxins, biliary obstruction, hemochromatosis, Wilson’s disease.
CIRRHOSIS

MANIFESTATIONS

- Ascites, edema, jaundice.
- Hepatosplenomegaly- thrombocytopenia.
- Portal hypertension- esophageal varices, caput medusae. See figure 40-15, Pg 938.
- Impaired production of clotting factors, albumin.
- Impaired metabolism of sex steroids (estrogen)- gynecomastia, palmar erythema, testicular atrophy, spider angiomata, telangiectasias.
- Encephalopathy- accumulation of ammonia.
CIRRHOSIS

- See text for details re portal hypertension, ascites, liver failure, etc.

- Death results from:
  1) Bleeding.
  2) Hepatic encephalopathy.
  3) Hepatorenal syndrome.
CHOLELITHIASIS

- The formation of stones in the gall bladder (the "chole cyst").
- Consist primarily of cholesterol, also bilirubin combined w/ calcium.
- Caused by:
  1) Change in the composition of bile.
  2) Bile stasis.
  3) Inflammation of the gall bladder.
CHOLELITHIASIS

- Seen in:
  1) Obesity. Also in rapid, massive weight loss.
  2) States of high estrogen - OCP’s, pregnancy, ERT.
- The 5 F’s:
  - Fat. Fair.
  - Female. Fourty. Fertile.
- BUT…. ANYONE can develop gall stones.
ACUTE CHOLECYSTITIS

- Associated w/ complete or partial biliary obstruction.
- Chemical irritation, inflammation, swelling, ischemia.
- Secondary bacterial infection.
- Infection can lead to gangrene and perforation.
ACUTE CHOLECYSTITIS

SYMPTOMS

- Biliary colic- RUQ / epigastric pain, radiating to the back or right shoulder (why the right shoulder?).
- Follows a fatty meal.
- Indigestion, vomiting, 25% have jaundice.
- Intensifying pain, fever as it progresses.
- Elevation of LFT’s- bilirubin, AST, ALT, alk phos.
CHRONIC CHOLECYSTITIS

- From chronic irritation by stones or repeated bouts of acute cholecystitis.
- Associated w/ acute exacerbations of inflammation, common duct stones, pancreatitis.
CHRONIC CHOLECYSTITIS

SYMPTOMS

- More vague than in acute cholecystitis.
- Fatty food intolerance, indigestion, post-prandial belching.
- Colicky pain (usually after a fatty meal).
- Fever often absent until infection sets in.
CHRONIC CHOLECYSTITIS

SYMPTOMS

- Liver enzyme / lab changes come and go, so you can’t hang your hat on one isolated panel.

- If pancreatitis is also present as the cause, the clinical picture will be further clouded by the superimposed symptoms of that.
CHOLELITHIASIS

DIAGNOSIS

- Ultrasound.
- Technetium scan.
- OCG- oral cholecystogram.
ACUTE PANCREATITIS

- Escape of pancreatic enzymes into the pancreas and surrounding tissue.
- Causes “autodigestion” of the pancreas.
- **Caused by:**
  1) Gallstones w/ obstruction.
  2) Alcohol.
  3) Hyperparathyroidism (and its hypercalcemia).
  4) Hyperlipidemia.
  5) Infection, drugs (thiazides, steroids).
ACUTE PANCREATITIS

RESULTS IN:

1) Fatty deposits in the abdominal cavity.
2) Hemorrhage, loss of fluids and electrolytes into the abdominal cavity in large amounts.
3) Hypocalcemia from precipitation of calcium in areas of fat necrosis.
ACUTE PANCREATITIS

**SYMPTOMS**

- Abrupt onset of severe epigastric pain, radiates to the back.
- Hypoactive bowel sounds.
- Tachycardia, hypotension, fever.

**LAB ABNORMALITIES**

- Elevated serum **amylase**, hypocalcemia, hypoglycemia, hyperbilirubinemia, leukocytosis, fall in hematocrit.
ACUTE PANCREATITIS

DIAGNOSIS

- Clinical presentation, lab.
- CT, ultrasound.
CHRONIC PANCREATITIS

2 EXCITING FLAVORS

- 1) CHRONIC CALCIFYING PANCREATITIS.
- 2) CHRONIC OBSTRUCTIVE PANCREATITIS.
CHRONIC PANCREATITIS

CHRONIC CALCIFYING PANCREATITIS
- Deposition of calcified “protein plugs” in the pancreatic ducts.
- Seen in alcoholics.

CHRONIC OBSTRUCTIVE PANCREATITIS
- Cystic fibrosis.
- Obstruction of the pancreatic duct by stenosis of the Sphincter of Oddi.
CHRONIC PANCREATITIS

SYMPTOMS

- Similar to acute pancreatitis but less severe.
- Progressive loss of the exocrine and endocrine function of the pancreas: diabetes, malabsorption.
CANCER OF THE PANCREAS

- 4th leading cause of cancer death in the U.S.
- Most have metastasized by the time of Dx.
- CAUSES:
  1) Smoking.
  2) Diet- high in fat, meat, salt, dehydrated and fried foods, refined sugars, soy beans, and nitrosamines.
- PROTECTED BY: diet high in fiber, Vitamin C, fruits and veggies, no preservatives
CANCER OF THE PANCREAS

SYMPTOMS

- Pain, jaundice, weight loss.
- Most are in the head of the pancreas → obstruction of the common bile duct → jaundice.

DIAGNOSIS

- Ultrasound, CT.