Caring for Patients with Common Health Problems of the Lower GI System

Module D

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Digestive System

Large Intestine

Structure of Colon

Large Intestine

- Vagal stimulation increases rhythmic contraction of proximal colon.
- Extrinsic parasympathetic fibers reach the distal colon through the pelvic nerves and can increase motility throughout the colon.

Large Intestine

- Movement of feces into the sigmoid colon and rectum stimulates the defecation reflex.
- Rectal wall stretches and the tonically constricted internal anal sphincter (smooth muscle with autonomic nervous system control) relaxes, creating the urge to defecate.

Gastrointestinal Bleeding

Upper GI Bleed

- Esophagus/Varices/Mallory Weiss Tear due to severe retching
- Stomach
- Duodenum
- Lower GI Bleed
  - Jejunum, Ileum, colon, rectum, cancer, hemorrhoids,

Presentation of GI Bleeding

- Hematemesis- Bloody vomitus
- Melena- Black, sticky, tarry foul smelling stools
- Hematochezia- Fresh bright red blood passed from the rectum
- Occult bleeding- Trace amounts of blood in normal appearing stools or gastric secretions

Continued

- Accumulation of blood is irritating and increases peristalsis, causing diarrhea
- Digestion of blood proteins originating from massive upper GI bleeding is reflected by increase in blood urea nitrogen (BUN).

Gastroesophageal Reflux Disease

Appendicitis

- Most common surgical emergency of the abdomen
- Affects 7-12% of population
- Common in ages 20-30, could develop at any age

Appendicitis

Continued

Diverticular disease

- Colonic herniations of mucosa through the muscle wall of the colon
- Usually in sigmoid of descending colon due to of increased pressures
- 3:1 higher incidence in females
- Usually asymptomatic ? 65% of older adults have it (this is diverticulosis)
Only 5% will develop diverticulitis

**Diverticulosis/Diverticulitis**

Diverticulitis

- Probably develops when a diverticula is perforated and becomes infected
- SXs ? LLQ colicky pain
- Fever
- Change in bowel habits (diarrhea, constipation
- May be difficult to diagnose in the elderly

**Diverticulitis**

- 1st sx may be confusion
- Joint pain
- BS poss. Hypoactive
- LLQ tenderness
- Rebound and guarding if peritonitis
- Palpable mass if abscess

**Diagnosis**

- Avoid sigmoidoscopy, BE and colonscopy in acute phase?? Perform after resolution
- CBC SMA
- CT if suspect abscess

**Treatment**

- If acute but mild
- Cipro 500mg or Septra bid x 10 d. &
- Flagyl 500 mg bid x 10 d.
- Liquid diet
- Follow up 2-3 days

**Treatment of Diverticulosis**

- Increased fiber such as bran or metamucil
- Use of antispasmodics such as Probanthine or Bentyl controversial

**Irritable Bowel Disease**

- Common chronic functional bowel disorder characterized by abdominal pain and alteration in bowel habits

- Affects 20 % of adult population

- Females > than males 30-50% of all GI visits

**Continued**

- Onset usually in late adolescence, early adulthood, sx persist for years
- Onset rare after 50 years

- Psychosocial factors contribute greatly and affect the clinical outcome

**Continued**

- Abdominal discomfort or pain of at least 12 weeks duration in the preceding 12 months
- 2 out of 3 of the following
  - Relief with defecation
  - Onset associated with change in the frequency of stool
  - Onset associated with change in the appearance of stool
Continued
- Defined according to predominant symptoms
  - Constipation
  - Diarrhea
  - Combination of both at alternating times

Continued
- + occasional diffuse tenderness with palpation, especially in LLQ
- CBC, sed rate, Diff
- Stool hemoccult
- Flex sigmoid
- Eval for lactose intolerance

Treatments
- Metamucil, bulking agents
- High fiber, Low fat diet
- Immodium (loperamide) for diarrhea
- Anti spasmodics
- Psychological support

Inflammatory Bowel Disease

Ulcerative Colitis

Ulcerative Colitis
- Diffuse inflammatory disease of bowel mucosa, usually distal colon and rectum, characterized most often by bloody diarrhea, increased urge to defecate with lower abdomen.
- Cramping can occur at any age but initial dx usually between 15-20 then another peak between 50-70.

Pathophysiology
- Severe in rectum and sigmoid colon.
- Inflammatory cytokines cause tissue damage.
- Small erosions form into ulcers, abscess formation, necrosis, and ragged ulceration of mucosa is evident.
- Edema and thickening of the lumen causes narrowing.

Symptoms
- Fever, elevated pulse rate
- Frequent diarrhea, urgency
- Bloody stools, crampy pain
- Dehydration, wt loss, anemia
- WBCs in stool
- May lead to colorectal CA, 10>x than general population

Diagnosis
- CBC, SMA, ESR
- Stool for hemoccult
- Stool for O & P if indicated
- Sigmoidoscopy, colonscopy with biopsy
- Barium Enema and x-ray films
- Lab values include low hemoglobin, hypoalbuminemia, and low serum K+.

Treatment
- Steroids- Supress the inflammatory response and alleviate the cramping pain.
- Broad Spectrum antibiotic may be prescribed if bacterial infection is suggested.
Severe disease can require hospital admission and administration of IV fluids.

**Crohn’s Disease**
- Chronic inflammatory disorder, usually beginning in early adulthood of granulomatous nature that may extend through the bowel wall layers.
- May occur anywhere from the buccal mucosa to distal right colon and anus.
- May be discontinuous. Occurs equally in both sexes, familial tendency may be +, increased incidence in eastern European Jews.

**Pathophysiology**
- Begins in intestinal submucosa and spreads across the intestinal wall.
- Creates a pattern called skip lesions.
- Ulcerations produce longitudinal and transverse fissures that extend inflammation into lymphoid tissue.
- Cobblestone projections of inflamed tissue surrounded by scarring.

**Clinical Manifestations**
- Diarrhea to decreased colonic absorption, bacterial overgrowth.
- Bile in colon that inhibits water absorption.
- Inflammation of the ileum causes tenderness in the lower right side of the abdomen.
- Wt loss, Vit B12 malabsorption, folic acid deficiency, Vit D, and calcium. Proteins may be lost leading to hypoalbuminemia.

**Treatment**
- Bulking agents
- Antibiotics
- Sulfasalazine
- Steroids
- Immunosuppressive agents
- Nutrition
- Surgery
- Increased risk of CA

**Hirschsprung Disease**
- Functional obstruction of the colon caused by inadequate motility.
- Exact cause unknown
- Increased in males and increased in the siblings of children with Hirschsprung Disease
- Increased in children with Down’s Syndrome

**Clinical Manifestations**
- Initial symptom usually constipation.
- Newborn may fail to pass meconium within 24 hours of birth.
- May repeatedly vomit yellow or green colored bile and may have a distended abdomen.
- Most serious complication in the neonatal period is enterocolitis due to fecal impaction.

**Evaluation and Treatment**
Definitive diagnosis rectal biopsy which shows an absence of ganglion cells in the submucosa of colon. X-ray films show dilated loops of colon, and contrast films (BE) show aganglionic areas. Infant may not expel the barium.

**Imperforate Anus**
- Congenital malformations of anorectal structures can obstruct the passage of feces.
- App. 40% of infants with anorectal malformations have other developmental anomalies.
- Imperforate anus may not be obvious.

**Intussusception**
- Telescoping or invagination of one portion of the intestine into another.
- Most frequent cause of acquired intestinal obstruction in infants.
- Usually the ileum invaginates the cecum and part of the ascending colon by collapsing through the ileocecal valve.

**Intussusception in Infants**
- Abdominal or anal “sausage”
- Blood from the rectum
  - Red currant jelly
- Colic: babies draw up their legs
- Distention
- Dehydration
- Emesis
- Face pale

**Continued**
- Most common, the proximal portion of the intestine, the intussusceptum, collapses into the distal portion, in the direction of peristaltic flow.

**Continued**

**Clinical Manifestations**
- Abdominal pain
- Irritable (colicky)
- Draws up knees
- Vomiting occurs soon after
- 60% of infants pass “current jelly” stools
- Most infants have a tender, sausage shaped abdominal mass
- Abdominal tenderness and distention develop as intestinal obstruction becomes more acute.

**Evaluation and Treatment**
- Ultrasound of abdomen
- Barium enema
- Hydrostatic pressure generated by air

**Diarrhea**
Increase in frequency of defecation and the fluidity and volume of feces.
Many factors determine stool volume and consistency, including water content of the colon and the presence of unabsorbed food, unabsorbable material, and intestinal secretions.
Normally 150 ml of water is excreted daily in the stool.

Continued
Large volume diarrhea caused by excessive amounts of water or secretions or both in the intestines.
Small volume diarrhea in which the volume of feces is not increased.

Osmotic Diarrheas
Non-absorbable substance in the intestine draws water into the lumen by osmosis.
Excess water and non-absorbable substance cause large volume diarrhea.

Secretory Diarrhea
Large volume diarrhea caused by excessive mucosal secretion of fluid and electrolytes or inhibition of Na, Cl, absorption.
Primary causes are bacterial enterotoxins, E. Coli, neoplasms.
Large volume diarrhea can result from excessive motility of the intestine.

Diarrhea’s
Always compare to previous normality
Establish increased frequency and loosening of stool
Acute vs. chronic

History
Duration
Fever
Blood
Quantity
Mucus
Cramps
N/V
Travel

Physical Exam
General Appearance
Wt loss

Presence of blood

Gastroenteritis
Viral GE
Short lived diarrhea
N & V, fever, cramps 2-3 days
No blood
Often epidemic

Travelers Diarrhea
50% caused by e.coli
Sudden profuse watery diarrhea caused by increased fluid excretion in intestinal wall
No blood

Invasive Diarrhea's
Shigella (Dysentary)(Gram neg.org.)
Invades the lumen of the intestine
Causes severe watery (possibly bloody) diarrhea
Fecal-oral, occ. Contaminated water
1-2 days incubation
Fever, malaise watery diarrhea, after 24 hrs. may progress to urgency, tenesmus, decreased volume freq stools

**Invasive Diarrhea’s**
Salmonella- Milder illness, incubation 1 day
Oral fecal
Ha, anorexia, fever, cramps, N & V, diarrhea? no blood
Duration 1-4 days to 3 weeks
Need stool, rectal culture

**Clinical Manifestations**
Dehydration
Electrolyte imbalance
Metabolic acidosis
Wt loss

**Pancreas**

**Pancreatitis**
Acute inflammation of the pancreas most frequently caused by excessive ETOH use, or gallstone blocking the sphincter of Oddi.
Acute pancreatitis is reversible, and can be resolved.
Chronic pancreatitis is irreversible and progressive.

**Frequency**
80% due to obstruction of biliary tree and alcoholism.

**Frequency**
20% due to
Drugs
Infection, hyperlipidemia
Structural abnormalities of the ducts
Surgery, vascular disease, and trauma

**Symptoms**
50% will have sudden, severe pain that radiates to back. Pain unrelieved by meds and lasts for days
N & V due to hypermotility or paralytic ileus secondary to the pancreatitis or peritonitis
Diaphoresis
Tachycardic, tachypneic
Temperature > 100 degrees, Leukocytosis
Hypoactive BS
Upper abd. tenderness

**Tests**
Serum amylase & lipase elevated on 1st day, return to normal on day 3-7
Plain films (supine and upright) may reveal calculi in area of sphincter of Oddi
US may show possible gallstones or dilatation of common hepatic duct
CT can visualize pancreas

68 **Prognosis**
- If necrosis of tissue from pancreatic enzymes 10-50% mortality
- Treatment ? supportive and in hospital

69 **Chronic Pancreatitis**
- Chronic alcohol abuse most common
- Causes continuous or intermittent abdominal pain, which usually intensifies after a meal
- Pain due to increased tissue pressure, ischemia, or ongoing injury
- Risk for narcotic addiction
- Occasionally manifestations of pancreatic enzyme deficiency

70 **Cancer of the Pancreas**
- Second most common GI cancer in US
- Males over age 75 have 10 x greater incidence than general population
  - Risk factors
    - Smoking, DM, high fat diet, coffee

71 **Signs and Symptoms**
- Insidious onset epigastric pain
- Weight loss
- Jaundice, nausea and vomiting
- Vague abdominal pain, back pain
- Very high mortality

72 **Objective**
- PE usually wnl early in disease
- Labs: mild anemia, hyperglycemia in 10-20%
- Elevated LFTs
- Serum amylase may be elevated
- Diagnosis usually made by CT or MRI

73 **Cholecystitis/Cholelithiasis**
- Acute or chronic inflammation of the gallbladder.
- Cholecystitis occurs when the cystic duct becomes obstructed, usually by a stone. In US 10% of men and 20% of women have gallstones by age 65? usually cholesterol stones

74 **Gallbladder**

75 **Cholelithiasis**
- Obstruction of gallstones
- May remain in gallbladder or be ejected with bile into the cystic duct
- If lodged in cystic duct will obstruct the flow of bile into and out of gallbladder causing inflammation

76 **Continued**
- Prevalent disorder incidence is 10-20%
- Gallstones are of two types: cholesterol and pigmented

| Cholesterol stones most common |

77 **Continued**

| Cholesterol gallstones form in bile that is supersaturated with cholesterol produced |
by the liver

**Risk factors**
- Obesity
- Weight loss (especially rapid)
- DM
- Cirrhosis
- Hyperlipidemia

**Signs and Symptoms**
- Abdominal pain and jaundice are the cardinal manifestations
- Nausea, vomiting, fever, malaise, aabd. Pain radiating to back
- Change in mental status or functional status
- Often PE unremarkable
- Dark urine
- Will require admittance

**Diagnosis**
- CBC, WBC may be elevated
- Serum bilirubin and alk phosphatase elevated if biliary obstruction present Abd.
- Ultrasound

**Stones**

**More Stones**

**Chronic Cholecystitis**
- Repeated mild attacks of acute cholecystitis, leading to thickened walls and shrinking of the gallbladder.
  - Eating fatty food will aggravate symptoms
  - Symptoms
  - Chronic indigestion
  - Vague abdominal pain
  - Nausea and belching

**Colorectal CA?? Risk factors**
- Age > 50
- High fat low fiber diet
- Inflammatory bowel disease
- + family hx of colorectal CA or gynecologic CA or multiple polyps Barrett’s esophagus
- Lower incidence in vegetarians

**Signs and Symptoms**
- Change in bowel habits abdominal pain, bloating, vomiting
- Low back pain or leg pain.
- Rectal bleeding Elderly may present with weakness or change in mental status

**Diagnosis**
- Colonoscopy!!!
  - Screening to catch early
  - FOBT ? ACS recommends every yr.
  - Sigmoidoscopy every 3-5 yrs
  - Once in a lifetime colonoscopy
Colonoscopy/Sigmoidoscopy
Barium Enema
Constipation
- Need to be able to detect any serious underlying illness and direct safe treatment.
- Decrease in # of bowel movements per week, hard stools, and difficult evacuation.
- Must be individually defined

Constipation
- Causes:
  - Megacolon (Enlarged or dilated colon)
  - Abdominal muscle weakness
  - Painful lesions
  - Low residue diet
  - Sedentary Lifestyle
  - Hypothyroidism
  - Diabetic Neuropathy

Mnemonic: CONSTIPATED
- Congenital: Hirschsprung disease
- Obstruction
- Neoplasms
- Stricture of colon
- Topical: painful hemorrhoids or fissure
- Impacted feces
- Prolapse of the rectum
- Anorexia and depression
- Temperature high, dehydration results
- Endocrine: hypothyroidism
- Diet, diverticulitis, and drugs

History
- Everything and anything may lead to constipation!
- Drugs
- Psych factors
- Other medical problems/treatments
- Chewing difficulties
- Self treatment to name a few

Objective

Management
- Fluids, fiber, environment
  - Stool softeners if hard stools
- Colace or DSS
- Glycerine suppositories ? 1 in AM

Features of Peritonitis
- Pain: front, back, sides, shoulders
- Electrolytes fall, shock ensues
Rigidity/rebound of anterior abdominal wall
Immobile abdomen & patient
Tenderness (rebound)
Obstruction
N/V
Increasing pulse, decreasing BP
Temperature falls, then rises
Increasing girth of abdomen
Silent abdomen (no bowel sounds)

Peritonitis

Liver
Largest organ in body
Covered by fibroelastic capsule called Glisson capsule
Capsule contains blood vessels, lymphatics, and nerves

Liver
Liver Lobes-small anatomic units
Hepatocytes-cells can regenerate
Lipocytes-store lipids, including Vit A
Sinusoids-receive venous and arterial blood, lined with Kupffer cells
Bile Canaliculi-conduct bile produced by hepatocytes and drain to common bile duct

Liver Continued
Secretion of Bile
700 to 1200 per day
Alkaline, bitter tasting, yellow green fluid
Contains bile salts, cholesterol, bilirubin, electrolytes, and water
Formed in hepatocytes and secreted into canaliculi
Bile salts required for the intestinal emulsification and absorption of fats

Metabolism of Bilirubin
Bilirubin-byproduct of destruction of aged red blood cells
Gives bile a greenish black color and produces the yellow tinge of jaundice
Aged RBC's are destroyed by Kupffer cells
Hemoglobin separated into component parts (heme-globin)

Liver (Vascular and Hematologic Functions)
Liver stores large amounts of blood
The amount stored at one time depends on pressure between arteries and veins
Kupffer cells remove bacteria and foreign particles from the portal blood
Synthesizes prothrombin, fibrinogen, and factors I, II, VII, IX, and X.

Metabolism of Nurtients
Fat is synthesized from carbs and protein primarily by liver
Fat enters liver as triglycerides, used to produce metabolic energy, or released in bloodstream as lipoproteins
Phospholipids and cholesterol are needed for hepatic production of bile salts, steroid hormones, components of plasma membranes, and other molecules.
Proteins
- Protein synthesis requires the presence of all the essential amino acids (obtained only from food)
- In hepatocytes, amino acids are converted to carbs by the removal of ammonia
- Ammonia converted to urea by the liver and passes into the blood to be excreted by the kidneys
- Liver also synthesizes nonessential amino acids and serum enzymes (AST, SGOT, ALT, SGPT, LDH)

Liver Continued
- Carbohydrates
- Metabolic Detoxification
- Storage of Minerals and Vitamins

Portal Hypertension
- Abnormally high blood pressure in the portal venous system
- Portal veins carry blood from the GI tract, pancreas, and spleen to the liver
- The portal veins, sinusoids, and hepatic veins compose the portal venous system

Pathophysiology
- Caused by disorders that obstruct or impede blood flow through any component of the portal venous system or vena cava

Continued
- Obstruction can occur due to
  - Liver thrombosis
  - Inflammation
  - Fibrosis of the sinusoids (Cirrhosis of liver, viral hepatitis)

Continued
- High pressure in the portal veins causes collateral vessels to open between the portal veins and systemic veins, in which blood pressure is considerably lower

Portal Hypertension

Complications
- Varices
- Splenomegaly
- Ascites
- Hepatic encephalopathy

Portal Hypertension

Venous Collaterals Abdominal Wall

Esophageal Varices
Ascites

Clinical Manifestations

Evaluation and Treatment
- Diagnosis made at time of variceal bleeding and confirmed by endoscopy and evaluation of portal venous pressure
- Distended collateral veins may radiate over the abdomen “Caput Medusae”
- Individual usually has hx of jaundice, hepatitis, or alcoholism

Continued
- Emergency management of bleeding varices includes compression of varices with an inflatable Sengstaken-Blakemore tube, which is shaped like a ballon with a bulb at the end

TIPPS Procedure

Ascites
- Accumulation of fluid in peritoneal cavity
- Traps body fluid “third spacing”
- Cirrhosis most common cause
- 25% of individuals who develop ascites caused by cirrhosis die within 1 year
- Continued drinking also

Pathophysiology
- Impaired excretion of sodium by kidneys promotes water retention
- Renal sodium retention stimulated by portal hypertension with intravascular hypervolemia and overflow in the peritoneal cavity
- Decrease in effective plasma volume, stimulating kidney to retain Na&H2O.
- Intravascular overload

Clinical Manifestations
- Wt gain
- Abdominal distention
- Increased abdominal girth
- Large volumes of fluid(10-20L) cause dyspnea by decreasing lung capacity
- 10% develop bacterial peritonitis due to paracentesis

Eval and Treatment
- Goal to relieve discomfort
- Dietary salt restriction
- Potassium sparing diuretics reduce ascites
- Lasix, Bumex
- Paracentesis

Hepatic Encephalopathy
- Neurologic syndrome
- Characterized by impaired cerebral function, flapping tremor (asterixis)
- EEG changes

Pathophysiology
- Liver dysfunction and collateral vessels that shunt blood around the liver to the
systemic circulation both permit toxins absorbed from the GI and circulates freely in the brain
End product of intestinal protein digestion, ammonia
Ammonia reaches brain and alters or interferes with neurotransmitters

126 Clinical Manifestations
Subtle changes include
- Personality
- Memory loss
- Lethargy
- Sleep disturbance
Symptoms then progress to
- Confusion
- Flapping tremor of the hands, convulsions, coma

127 Eval and Treatment
- No specific diagnostic test
- EEG, blood tests
- Correction of fluid and electrolyte imbalances and withdrawal of depressant drugs
  first step of treatment
- Reduction of blood ammonia major objective

128 Jaundice
- Icterus-yellow or greenish pigmentation of the skin caused by hyperbilirubinemia
- Excessive hemolysis of RBC’s disorders of bile ducts or liver cells

129 Pathophysiology
- Extrahepatic Obstructive Jaundice
- Intrahepatic Obstructive Jaundice
- Hemolytic Jaundice

130 Clinical Manifestations
- Urine darken before onset of jaundice
- Complete obstruction of bile flow from the liver to the duodenum causes light colored stools
- Fever, chills, and pain accompany jaundice
- Anorexia, malaise, fatigue
- Yellow discoloration to sclera and then skin
- Itching (elevation of alkaline phos and bili accumulation of skin)

131 Eval and Treatment
- Eval of serum (conjugated or unconjugated bili)
- Look at history and physical examination
- Underlying disorders

132 Hepatorenal Syndrome
- Consists of advanced liver disease and functional renal failure with oliguria, NA &H2O retention, hypotension, and peripheral vasodilation
- Caused by circulatory alterations

133 Pathophysiology
- Oliguric hepatic failure generally accompanies a sudden decrease in blood volume secondary to massive GI bleed, or hypotension due to liver failure
Excessive diuretics to treat ascites

134 **Clinical Manifestations**
- Oliguria
- Jaundice
- Ascites
- GI bleed
- Systolic BP below 100 mm Hg
- Anorexia
- Weakness
- Fatigue

135 **Eval and Treatment**
- Potassium elevated in terminal stages of illness
- BUN/Creat increase
- Urine NA concentrations below normal

136 **Liver**

137 **Cirrhosis**
- Leading cause of death in US
- Diffuse fibrosis and nodules
- Liver firm or hard when palpated
- Structural changes result from fibrosis and inflammation
- Biliary channels may be altered or obstructed producing jaundice

138 **Ascites**

139 **Continued**

140 **Caput Medusa**

141 **Spider Angiomata**

142 **Hernia**

143 **Jaundice**

144 **ICTERUS**

145 **Pitting Edema**

146 **Viral Hepatitis**
- Affects the liver
- Five strains of viruses cause different hepatitis
  - Hep A
  - Hep B
  - Hep C
  - Hep D
  - Hep E

147 **Hepatitis A**
- Feces
- Bile
Usual transmission is fecal-oral route (contaminated food and water), virus can be spread also by transfusion of infected blood
45% of adults in urban areas have HAV antibodies in their blood

Incubation period (time between exposure and onset of symptoms) for hepatitis A is 4 to 6 weeks
Fecal shedding of the virus is greatest 10-14 days before onset of symptoms and during first week of symptoms
Most contagious during this time

Hepatitis B
Transmitted through contact with infected blood, body fluids, or contaminated needles
Sexually transmitted
Transmission among homosexual men
People receiving hemodialysis, multiple transfusions, immunosuppressive drugs (greater risk of exposure)
Incubation period of 6 to 8 weeks

Hepatitis C
Caused by posttransfusion
Hep C antibody present in 44% of blood donors
Greater risk for those who need large volumes of blood replacement and IV drug users
FDA has approved for assay for screening Hep C antibodies in blood products

Hepatitis D
Occurs in individuals with Hep B
The virus depends on Hep B virus for replication
Parental drug users high incidence
The clinical course of HDV is similar to that of hepatitis A and B, could be more severe

Hepatitis E
Most common in developing countries and is transmitted by the fecal oral route, usually by contaminated water
Highest mortality in pregnant women

Pathophysiology
Hepatic cell necrosis, scarring
Kupffer cell hyperplasia
Infiltration by mononuclear phagocytes
Cellular injury by cell mediated immune mechanisms (cytotoxic T cells and natural killer cells)

Continued
Regeneration of hepatic cells begins within 48 hrs of injury
Inflammation can damage or obstruct bile canaliculi, leading to cholestasis and obstructive jaundice
Damage more severe with HepB and Hep C

Clinical Manifestations
Acute viral hepatitis causes abnormal liver function test results
AST, ALT are elevated
Clinical course of hepatitis consists of three phases: prodromal, icteric, and recovery phase

Prodromal Phase
Begins 2 weeks after exposure and ends with appearance of jaundice
Fatigue, anorexia, malaise, nausea, vomiting, ha, cough, and low grade fever
RT upper abdominal pain
Wt loss of 2 to 4 kg is not unusual
Infection highly transmissible during this phase

**Icteric Phase (Jaundice)**
- Begins 1 to 2 weeks after the prodromal phase and lasts 2 to 6 weeks
- Hepatocellular destruction and intrahepatic bile stasis cause jaundice
- Urine dark
- Stools clay colored before onset of jaundice
- Prothrombin time may be prolonged

**Recovery Phase**
- Begins with resolution of jaundice (6 to 8 weeks after exposure)
- LFT’s return to normal within 2 to 12 weeks after the onset of jaundice
- Chronic Hep may begin at this point
- Persistent liver inflammation after HepB, HepC, HepD. LFT’s remain abnormal for longer than 6 months

**Eval and Treatment**
- Most specific diagnostic test for viral hepatitis is serologic analysis
- No specific treatment for acute viral hepatitis
- Physical activity restricted
- Low-fat, high carb diet
- Interferon can be useful in tx of chronic HepB and HepC

**Continued**
- No direct contact with blood or body fluids of patients with HepB or HepC
- Administration of immunoglobulin before exposure or early in the incubation period can prevent HepA
- Vaccine for HepA and Hep B available
- Prophylactic immunoglobulin administered before exposure can also prevent HepB

**Surgically Induced Alterations in the Structure of the Bowel**
- When alterations in bowel elimination become life threatening medical management fails, surgical intervention is necessary
- Diversionary surgical procedures of the bowel include ileostomy, cecostomy, and colostomy

**Ileostomy**

**Ileostomy Pouch**

**Cecostomy**

**Colostomy**

**Indications for Colostomy**