

The Patient with Digestive Disorders

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Stomach Disorders

- Gastritis—inflammation of the gastric mucosa
- Can be erosive or nonerosive
- Role of prostaglandins
- With progressive disease, stomach lining thins, parietal cell functioning becomes compromised and the patient will develop *pernicious anemia*
- Increased risk of cancer

Gastritis continued

- Onset of infection with *H.pylori* can result in gastritis
- Other pathogens implicated are CMV (in HIV patients), staph, strep, E.coli or salmonella
- NSAIDS
- Ingestion of corrosive substances
- radiation

Chronic Gastritis

- Type A has autoimmune pathogenesis, genetically linked
- Type B is caused by *H. pylori*. Direct correlation between number of organisms and degree of cellular abnormality.
- Can also be caused by alcohol ingestion, radiation therapy and smoking. Other causation may be uremia.

Physical Manifestations

- Abdominal tenderness
- Bloating
- Hematemesis
- Melena
- Can progress to shock

Interventions

- Treat symptomatically
- Remove causative agents
- Treat *H. pylori* with bismuth, amoxicillin and Flagyl
- Treat with H₂ receptor antagonists to block gastric secretions
- Antacids as buffers
- May need B12
- Instruct patient about medications that exacerbate the problem such as steroids, NSAIDS, ASA, erythromycin and chemotherapeutic agents

Diet Therapy

- Avoid known foods that cause S/S
- Tea, coffee, cola, chocolate, mustard, paprika, cloves, pepper and hot spices may cause discomfort

Management cont.

- Surgery--Partial gastrectomy, pyloroplasty, vagotomy or even total gastrectomy may be indicated
- Stress reduction

Peptic Ulcer Disease

- Peptic ulcer is a mucosal lesion of the stomach or duodenum
- Results when gastric mucosal defenses become impaired and no longer protect the epithelium
- Gastromucosal prostaglandins increase the barrier against acid
- Gastric Ulcers can be caused by reflux of bile into the stomach, by delayed emptying of stomach resulting in backflow of duodenal contents; decreased blood flow also will alter the protective barrier

Peptic Ulcers continued

- Duodenal Ulcers—95% develop in the first portion of the duodenum
- Characteristic feature of a duodenal ulcer is high gastric acid secretion
- Protein rich meals, calcium and vagal excitation stimulate acid secretion
- Up to 95% to 100% of clients with duodenal ulcer disease have H.pylori
- This pathogen produces substances that damage the gastric mucosa
- Urease produced contributes further to the breakdown.

Stress Ulcers

- Acute gastric mucosal lesions occurring after an acute medical crisis
- Associated with HI, burns, respiratory failure, shock, and sepsis.
- Multifocal lesions occur in proximal stomach and duodenum
- Begin as focal areas of ischemia and may progress to massive hemorrhage

Complications of ulcers

- Hemorrhage in 15-25% of clients
- Perforation—severe pain will ensue. Abdomen is tender, rigid, and boardlike and the client will assume the knee-chest position to decrease abdominal wall tension-----is a surgical emergency
- Pyloric obstruction—caused by scarring, edema, inflammation or a combination of these

Distinguishing between gastric and duodenal ulcers

Gastric ulcers

1. Usually in those 50 yrs. And older
2. Equal proportion of males to female
3. Blood group not defining
4. May be malnourished
5. Normal or hyposecretion of stomach acid
6. Heal and recur
7. Pain occurs after a meal
8. Heals and recurs in same area
9. Atrophic gastritis

Duodenal Ulcers

1. Occur in those 40-50 yo
2. Equal male/female ratio
3. Most often type O blood
4. Well nourished
5. Hypersecretion of stomach acid
6. Occurs 90 min. to 3 hrs. after meal
7. Eating relieves pain. Melena more common than hematemesis
8. No gastritis

Diagnostic Testing

- EGD
- H.pylori testing—by breath test, serologic testing (antibodies revealed). Antibody testing can not be used to determine eradication.

Drug Therapy

- Antisecretory drugs such as Prilosec, Prevacid, Aciphex, Nexium
- H₂ receptor antagonists such as Pepcid, Zantac, Axid, Tagamet
- Prostaglandin analogs such as Cytotec. Actually enhances the mucosal resistance
- Antacids—buffer and prevent the formation of pepsin. Mylanta and Maalox are examples (aluminum and magnesium hydroxide). Be careful if CHF. Tums is calcium carbonate which actually triggers gastrin release.....rebound secretion.
- Antacids may interfere with certain medications such as Dilantin, ketoconazole and tetracycline.

Drug Therapy cont.

- Mucosal barrier fortifiers such as carafate.
- Creates a protective coat

Diet Therapy

- Bland diet may be helpful
- Food itself acts as an antacid
- Avoid caffeine
- Avoid both decaffeinated and caffeinated coffee because coffee causes stimulation of gastrin
- Avoid bedtime snacks which increase secretion of acid
- Eat small regular meals

Nonsurgical Management

- Treat hypovolemia
- Recognize s/s of hypovolemia
- Ready patient for endoscopy
- Saline lavage
- NG tube placement
- Acid suppression
- Monitor and document character of stools
- Avoid anti-inflammatories
- Administer blood products

Surgical Management

- Used to:

Reduce the acid-secreting ability of the stomach

Treat patients who do not respond to medical therapy

Treat a surgical emergency that develops as a complication of PUD

Surgical procedures

- Gastroenterostomy—permits neutralization of gastric acid by regurgitation of alkaline duodenal contents into the stomach. Also will perform vagotomy to decrease vagal influences
- Vagotomy—eliminates the acid-secreting stimulus to gastric cells and decreases the responsiveness of parietal cells. Can be selective, truncal or proximal.
- Pyloroplasty—widens the exit of the pylorus

Complications associated with partial gastrectomy

- Deficiency of B12
- Folic acid
- Iron
- Impaired calcium absorption
- Reduced absorption of vitamin D
- Result of shortage of intrinsic factor and the now rapid entry of food into the bowel which decreases absorption
- Nurse should monitor CBC, assessment of tongue for atrophic glossitis, s/s of anemia

Irritable Bowel Syndrome

- Most common digestive disorder seen in clinical practice
- Characterized by the presence of diarrhea, constipation, and abdominal pain and bloating
- Believed to be due to impairment in the motor or sensory function of the GI tract
- Cause unknown
- Dx made by careful history, labs and dx procedures which exclude more serious conditions

Irritable Bowel

- Food intolerances may be associated with IBS
- Dairy products and grains can contribute to bloating, flatulence and distention
- Occurs 2:1 more often in women
- Education is cornerstone of treatment
- Drug therapy includes fiber, tricyclic antidepressants, antidiarrheal agents, laxatives and anticholinergics
- Stress management may be helpful

Irritable Bowel

- Avoid caffeine, alcohol, beverages that contain sorbitol or fructose.

Neoplastic Disorders

Background

- 1. Large intestine and rectum most common GI site affected by cancer
- 2. Colon cancer is second leading cause of death from cancer in U.S.

B. Client with Polyps

1. Definition

- a. Polyp is mass of tissue arising from bowel wall and protruding into lumen
- b. Most often occur in sigmoid and rectum
- c. 30% of people over 50 have polyps

Neoplastic Disorders

Pathophysiology

- a. Most polyps are adenomas, benign but considered premalignant; < 1% become malignant but all colorectal cancers arise from these polyps
- b. Polyp types include tubular, villous, or tubularvillous
- c. Familial polyposis is uncommon autosomal dominant genetic disorder with hundreds of adenomatous polyps throughout large intestine; untreated, near 100% malignancy by age 40

Client with Polyps

Manifestations

- a. Most asymptomatic
- b. Intermittent painless rectal bleeding is most common presenting symptom

Collaborative Care

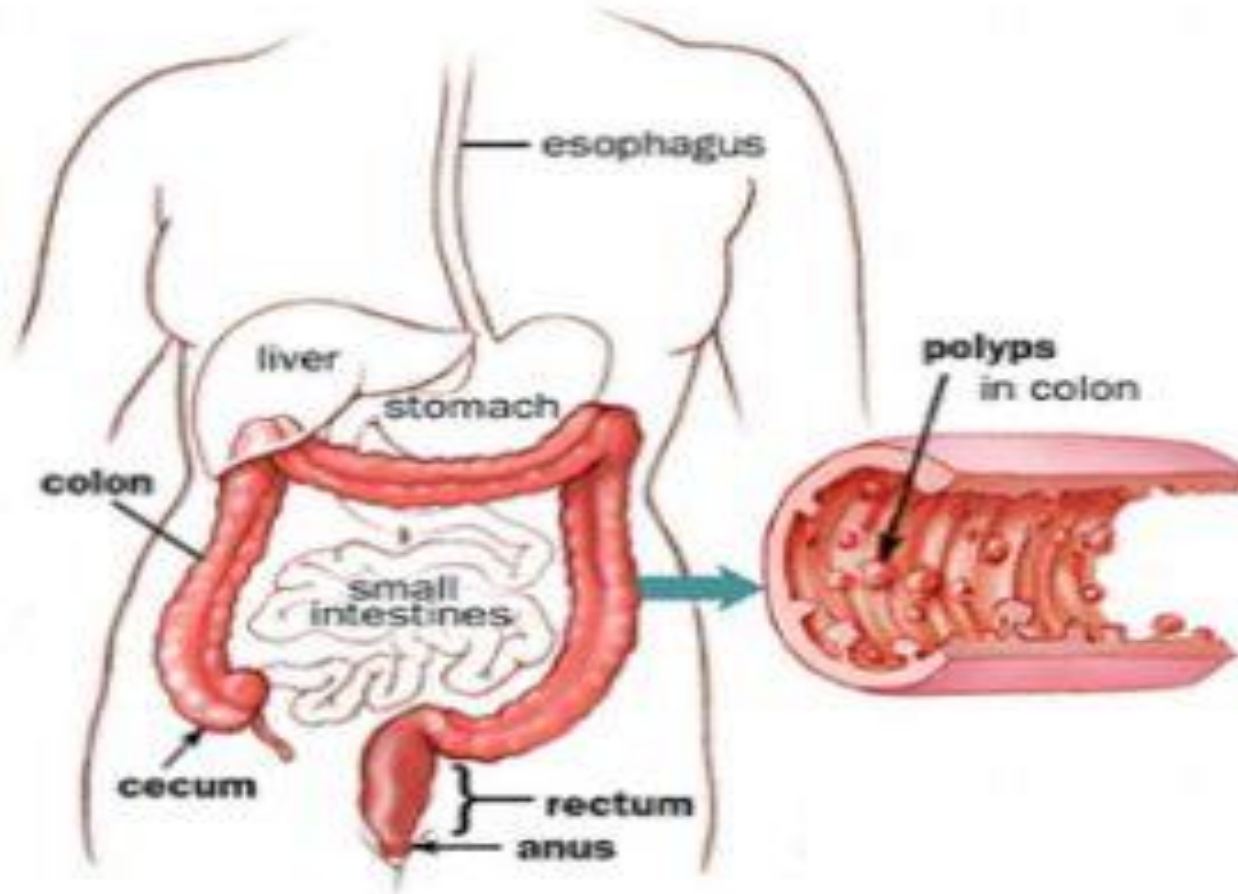
- a. Diagnosis is based on colonoscopy
- b. Most reliable since allows inspection of entire colon with biopsy or polypectomy if indicated
- c. Repeat every 3 years since polyps recur

Client with Polyps

Nursing Care

- a. All clients advised to have screening colonoscopy at age 50 and every 5 years thereafter (polyps need 5 years of growth for significant malignancy)
- b. Bowel preparation ordered prior to colonoscopy with cathartics and/or enemas

Polyps



This is what polyps look like inside the colon

Client with Diverticular Disease

Definition

- a. Diverticula are saclike projections of mucosa through muscular layer of colon mainly in sigmoid colon
- b. Incidence increases with age; less than a third of persons with diverticulosis develop symptoms

Risk Factors

- a. Cultural changes in western world with diet of highly refined and fiber-deficient foods
- b. Decreased activity levels
- c. Postponement of defecation

Client with Diverticular Disease

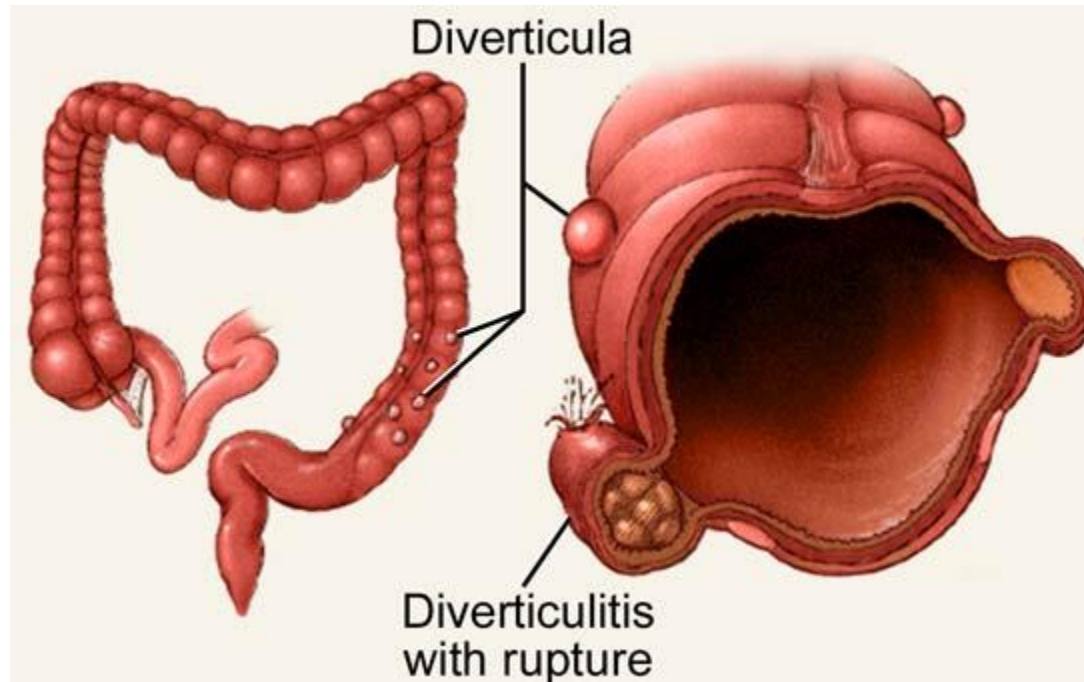
Pathophysiology

- a. Diverticulosis is the presence of diverticula which form due to increased pressure within bowel lumen causing bowel mucosa to herniate through defects in colon wall, causing outpouchings
- b. Muscle in bowel wall thickens narrowing bowel lumen and increasing intraluminal pressure
- c. Complications of diverticulosis include hemorrhage and diverticulitis, the inflammation of the diverticular sac

Clients with Diverticular Disease

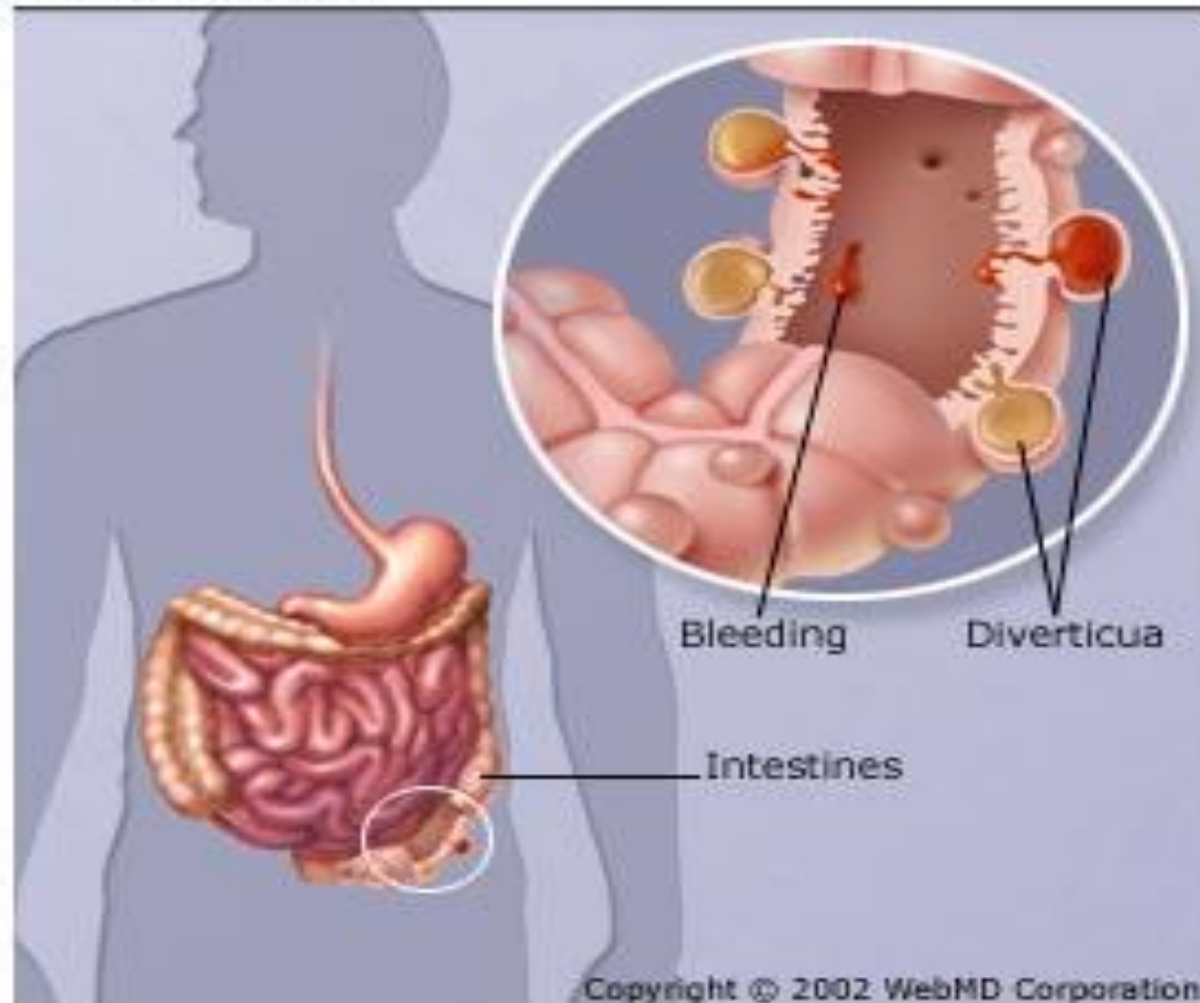
- d. Diverticulitis: diverticulum in sigmoid colon irritated with undigested food and bacteria forming a hard mass (fecalith) that impairs blood supply leading to perforation
- e. With microscopic perforation, inflammation is localized; more extensive perforation may lead to peritonitis or abscess formation

Diverticulitis



Diverticulitis

Diverticulitis



Client with Diverticular Disease

Manifestations

- a. Pain, left-sided, mild to moderate and cramping or steady
- b. Constipation or frequency of defecation
- c. May also have nausea, vomiting, low-grade fever, abdominal distention, tenderness and palpable LLQ mass
- d. Older adult may have vague abdominal pain

Complications

- a. Peritonitis
- b. Abscess formation
- c. Bowel obstruction
- d. Fistula formation
- e. Hemorrhage

Client with Diverticular Disease

Collaborative Care: Focus is on management of symptoms and complications

Diagnostic Tests

- a. Abdominal Xray: detection of free air with perforation, location of abscess, fistula
- b. Barium enema contraindicated in early diverticulitis due to risk of barium leakage into peritoneal cavity, but will confirm diverticulosis
- c. Abdominal CT scan, sigmoidoscopy or colonoscopy used in diagnosis of diverticulosis
- d. WBC count with differential: leukocytosis with shift to left in diverticulitis
- e. Hemocult or guiac testing: determine presence of occult blood

Client with Diverticular Disease

Medications

- a. Broad spectrum antibiotics against gram negative and anaerobic bacteria to treat acute diverticulitis, oral or intravenous route depending on severity of symptoms
 - Flagyl plus Bactrim or Cipro
- b. Analgesics for pain (non-narcotic)
- c. Fluids to correct dehydration
- d. Stool softener but not cathartic may be prescribed (nothing to increase pressure within bowel)
- e. Anticholinergics to decrease intestinal hypermotility

Clients with Diverticular Disease

Dietary Management

- a. Diet modification may decrease risk of complications
- b. High-fiber diet (bran, commercial bulk-forming products such as psyllium seed (Metamucil) or methycellulose)
- c. Some clients advised against foods with small seeds which could obstruct diverticula

Client with Diverticular Disease

Treatment for acute episode of diverticulitis

- a. Client initially NPO with intravenous fluids (possibly TPN)
- b. As symptoms subside reintroduce food: clear liquid diet, to soft, low-roughage diet psyllium seed products to soften stool and increase bulk
- c. High fiber diet is resumed after full recovery

Surgery

- a. Surgical intervention indicated for clients with generalized peritonitis or abscess that does not respond to treatment
- b. With acute infection, 2 stage Hartman procedure done with temporary colostomy; re-anastomosis performed 2 – 3 months later

Client with Diverticular Disease

Nursing Care: Health promotion includes teaching high-fiber foods in diet generally, may be contraindicated for persons with known conditions

Nursing Diagnoses

- a. Impaired Tissue Integrity, gastrointestinal
- b. Pain
- c. Anxiety, related to unknown outcome of treatment, possible surgery

Home Care

- a. Teaching regarding prescribed diet, fluid intake, medications
- b. Referral for home health care agency, if new colostomy client

Colorectal Cancer

- Familial
- Autosomal dominant inherited genetic disorder known as *familial adenomatous polyposis*
- More prevalent after age 50
- Can metastasize by direct invasion or by migrating via the blood or lymph
- Risk factors include genetics, dietary habits (animal fat, decreased bowel transit time, low fiber diets) and the occurrence of inflammatory bowel disease

Colorectal cancer

- Generally are adenocarcinomas
- Multistep process resulting in a number of molecular changes including loss of tumor suppressor genes and activation of oncogenes that alter colonic mucosa cell division.
Proliferation of colonic mucosa forms polyps that can be transformed to malignant tumors. Adenomatous polyps.

Colorectal Cancer

- Tumors predominantly develop in the sigmoid colon or rectum
- Colon tumors can be spread by peritoneal seeding during surgical excision
- Tumors can cause bowel obstructions
- 20% are diagnosed at time of emergency hospitalization for bowel obstruction
- 75% of all colorectal cancers have no known predisposing cause
- Inflammatory bowel diseases may pose an increased risk for tumor development

Colorectal Cancer

- Manifestations—rectal bleeding, anemia and a change in the character of the stool
- Lab assessment—CBC, elevated liver enzymes, +fecal occult blood test (Ensure that the patient is not on NSAIDS). Two separate stool samples should be tested on 3 consecutive days
- CEA (carcinoembryonic antigen) may be elevated in 70% of people with colorectal cancer
- Colonoscopy, sigmoidoscopy may help reveal polyps
- Liver scan may locate distant sites of metastasis

Colorectal Cancer

- Genetic counseling may be appropriate so careful history is very important
- Classified by Dukes' staging
 1. Stage A indicates that the tumor has penetrated into the bowel wall
 2. Stage B- the tumor has penetrated through the bowel wall
 3. Stage C-bowel wall penetration w/involvement of the lymph
 4. Stage D-mets

Colorectal Cancer

- Radiation has not improved outcomes except in regional disease affecting the rectum
- Chemotherapy has proven efficacious, especially in Stages B & C. Drug of choice is 5-FU. Side effects include diarrhea, mucositis and skin effects. Trying other medications such as leukovorin and irinotecan. Studies for use of monoclonal antibodies are under way.

Colorectal Cancer

- Surgical Management
- Three most common surgeries are:
 1. **Hemicolectomy**—excision of the involved area of the colon with reanastomosis
 2. **Colon resection**—if healing is thought to be in jeopardy, a colostomy will be created
 3. **Abdominoperineal resection**—indicated when rectal tumors are present. This approach generally requires a permanent colostomy.

Colostomy—Nursing Role

- Apply pouch system
- Assess stoma. Should appear pink and moist.
- Nurse reports any of the following:
 1. Signs of ischemia.....
 2. Unusual bleeding
 3. Mucocutaneous separation
 4. Signs of leakage

Colostomy Care

- Teach patients and families:
 1. Normal appearance of the stoma
 2. Signs and symptoms of complications
 3. Measurement of the stoma
 4. Care and application of the appliance
 5. Measures to protect the skin
 6. Dietary measures to control gas and odor
 7. Resumption of activities

Colostomy

- Gas producing foods include broccoli, asparagus, cucumbers, brussels sprouts, cabbage, cauliflower, garlic, turnips and beer
- Crackers, toast and yogurt can help prevent gas

Intestinal Obstruction–mechanical

- Mechanical obstruction can be caused from adhesions, tumors, hernias, fecal impactions, strictures, and vascular disorders
- Regardless of age, *adhesions* are the most common cause of mechanical obstruction.
- Adhesions are bands of granulation and scar tissue that develop as a result of inflammation, encircle the intestine and constrict its lumen

Intestinal Obstruction

- *Paralytic ileus* is a nonmechanical obstruction caused by physiologic, neurogenic, or chemical imbalances associated with decreased peristalsis either from trauma or from toxins
- Number of causative factors including MI, hypokalemia, vascular insufficiency, shock, or peritonitis. Any diffuse inflammatory response can cause this problem.

Intestinal Obstruction—Paralytic Ileus

- May have vomiting
- Constant, diffuse discomfort
- Diarrhea may be present in partial obstruction
- Abdominal distention will be present
- Abdominal rigidity
- Borborygmi which progresses to a quiet abdomen

Bowel Obstruction

- No definitive lab test
- CT useful
- Flat-plate and upright films will reveal distention of loops of intestine with fluid and gas in the small intestine and with the absence of gas in the colon
- Presence of free air in the abdomen indicates perforation

Paralytic Ileus Care

- NPO
- NG tube or nasointestinal tube (cantor, Miller-Abbott) for decompression
- Enemas may be helpful
- Fluid and electrolyte replacement are important
- Ice chips sparingly, not lemon glycerine swabs
- Closely monitor I&O
- Opioids may be withheld
- Mobilize patient if possible

Peritonitis

- Acute inflammation of the endothelial lining of the abdominal cavity or peritoneum. Is a life-threatening illness.
- If peritoneal cavity is contaminated by bacteria, the body produces an inflammatory reaction that walls off a localized area to fight the infection. This reaction involves vascular dilation and increased capillary permeability locally. If this local walling off is not effective, the inflammation spreads and becomes *peritonitis*

Peritonitis–pathophysiology

1. Vascular dilation continues,
2. extra blood is brought to the area of inflammation
3. Fluid is shifted from the ECF compartment into the peritoneal cavity resulting in “third spacing”.
4. This shifting then will affect circulatory volume.
5. Hypoperfusion of kidneys can result
6. Peristalsis will slow
7. Bowel lumen will become distended with gas and fluid
8. Resp. problems can ensue

Causes of peritonitis

- Appendicitis
- PUD
- Diverticulitis
- Gangrenous gallbladder
- Bowel obstruction
- Secondary to CAPD
- Ulcerative colitis

Presentation of Peritonitis

- Abdominal pain and tenderness which may be referred to the chest or shoulder—these are the cardinal signs
- Distended abdomen
- Nausea, vomiting and anorexia
- Diminished bowel sounds
- Rebound tenderness
- High fever
- Tachycardia
- Dehydration
- Decreased urinary output
- Possible respiratory compromise

Diagnostics

- Elevated WBCs with a shift to the left (bands)
- Possible positive blood cultures
- Abdominal xray will reveal free air or fluid—and edema
- Peritoneal lavage will reveal more than 500 WBCs/mL³ of fluid, greater than 50,000 RBCs/mL or the presence of bacteria on a gram stain

Interventions for Peritonitis

- Hospitalized
- IV fluids and antibiotics
- Daily weight
- I&O
- NG tube
- O2
- Pain medications
- Possible exploratory lap once stabilized
- Wound healing by secondary intention w/packing and irrigation

Peritonitis—care continued

- Upon discharge, teach patient
 1. Proper handwashing
 2. Wound care—may require home health nurse
 3. Reporting fever, unusual drainage or swelling, redness or bleeding from the incision site
 4. Presence of abdominal unlike experienced upon discharge.....

Chronic Inflammatory Bowel Disease

Ulcerative colitis

- Characterized by diffuse inflammation of the intestinal mucosa
- Result is loss of surface epithelium with ulceration and abscess formation
- Usually begins in rectum and continues gradually and progressively toward the cecum
- Fibrosis and retraction of the bowel result
- Genetically linked autoimmune in nature—seen more commonly in European Jews and Ashkenazic Jews

Complications of ulcerative colitis

- Perforation
- Toxic megacolon
- Hemorrhage
- Abscesses
- Increased likelihood of cancer
- obstruction

- Causes tenesmus (uncontrollable straining)
- Diarrhea
- Loss of vital nutrients
- May lead to malnutrition

Diagnosis

- Elevated ESR
- Increased WBC
- r/o ova and parasites
- Barium enema will be definitive
- Sigmoidoscopy is most definitive diagnostic procedure

Treatment

- Antidiarrheals
- Salicylate compounds (sulfasalazine) acts by affecting prostaglandins
- Steroids
- Immunosuppressive drugs—alone not helpful but if used in combination with steroids—positive outcomes
- Diet—NPO, TPN, low-fiber diet (debatable), avoid lactose containing foods
- Surgery—possible proctocolectomy with ileostomy or total colectomy with a continent ileostomy
- Psychosocial support—support groups

Nursing Care of Clients with Bowel Disorders

Factors affecting bodily function of elimination

A. GI tract

- 1. Food intake
- 2. Bacterial flora in bowel

B. Indirect

- 1. Psychologic stress
- 2. Voluntary postponement of defecation

C. Normal bowel elimination pattern

- 1. Varies with the individual
- 2. 2 – 3 times daily to 3 stools per week

Nursing Diagnosis for Patients with Digestive Disorders

- Risk for infection (septicemia)
- Deficient fluid volume (mixed)
- Acute pain
- Risk for imbalanced nutrition, less than body requirements
- Anxiety or fear (specify level)
- Knowledge deficient (learning need) regarding condition, prognosis, treatment regimen, self-care, and discharge needs
- Risk for infection
- Acute pain
- Knowledge deficient (learning need) regarding condition, prognosis, treatment regimen, self-care, and discharge needs