Clinical Pathology Conference
45 yo female with toxic megacolon

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Learning objectives

1) Define toxic megacolon.

2) Discuss common clinical scenarios that predispose a patient to develop toxic megacolon.

3) Discuss the differential diagnosis of toxic megacolon and the usual work-up of these cases.
History of Present Illness:

• 45 yowf presents to PCP for third visit in 14 days for abdominal pain and diarrhea.
• First presentation 2 weeks prior with abdominal cramping, nausea, vomiting.
• Treatment: Imodium, Tylenol and ibuprofen.
HPI:

• She re-presented 3 days later later with persistent diarrhea (8-10 “brown water” bowel movements a day with abdominal cramping), electrolytes were ordered.

• She was instructed to drink Gatorade, take Lomotil.

• She called 2 days later to state that Lomotil was not working and a clinic appointment was made for 4 days in future and stool studies were ordered.
HPI:

• Third presentation: Increased abdominal girth, persistent diarrhea and abdominal pain.

• Pertinent negative: fever, chills, recent travel, sick contacts, hematochezia, melena, rashes, CP, SOB, urinary symptoms.

• She was able to keep down liquids and had not attempted to eat solids x 24 hours.
Past Medical History:

- Hyperlipidemia
- Hypertension
- Obesity
- No previous surgeries
Medications

- Lomotil
- HCTZ
- Atenolol
- Gemfibrozil
- ASA
- Tylenol
- Vit E
- Multivitamins
- Colace (on hold)
- Calcium
- Vit D
Past history:

• Social- No history of tobacco, drugs or alcohol abuse. Single. Works at S&W in no-patient contact area.

• Allergies- None

• FH- Mother is HTN, DM II and Hyperlipidemia.

• Father- deceased at age 43 sec to MI.
Physical Exam:

- Vital signs: BP-136/68, HR-110, T-102.1, O2 Sat - 97% on room air.

- Gen: Alert, interactive, talkative, coherent, supine without respiratory distress with obviously grossly distended abdomen.

- CV: RR no M/G/R

- Resp: CTA B- no W/R/R
Physical Exam:

- Abdomen- distended, diffusely tender, voluntary guarding, no rebound, + tympanic, + high pitched bowel sounds

- Ext- no rashes, no edema

- Rectal exam?
Laboratory:

- Sodium 128
- potassium 3.5
- chloride 90
- bicarbonate 25
- BUN 19
- Creatinine 1.2
- WBC 11.6 (77% granulocytes)
- hemoglobin 10.8
- platelets 779,000

- INR 1.5
- lactic acid 1.5
- T Bili 1.1
- alk phos 183
- AST 28
- ALT 20
- albumin 1.5
- magnesium 1.9
- calcium 8.1
- phosphate 2.7
Laboratory:

- Stool for C diff x 1- negative.

- Urgent CT abdomen- Markedly dilated transverse colon of 7.6 cm. Several dilated loops of small bowel without a distinct transition zone. No pneumatosis, free air or free fluid. Mural thickening of the ascending colon near the hepatic flexure.
Supportive care was provided and a diagnosis was made after several investigations occurred.
Who gets the workup?

- Investigation is usually unnecessary for patients presenting within 24 hours of the onset of the diarrhea.
- Fever
- Blood/Pus in stools
- Recent antibiotics use
- Immunosuppressed patients, HIV/AIDS
- History of inflammatory bowel disease
- Travel to endemic areas
- Work exposures (food handling, daycare, veterinarian)
- Anal intercourse
Important questions for the workup of patients with diarrhea

- Usual pattern of BM
- Current pattern
- Volume of stools
- Nocturnal diarrhea
- Blood/Mucus
- Hx of Travel
- Dietary changes
- Exacerbation of diarrhea by different foods
- Drugs/medications use (laxatives, antibiotics)
- Change in the nature of food intake (diet products, pureed, liquids, etc)
- Hx of surgeries
- Rectal exam
- Work exposure (patients, handling food?)
Workup

• Fecal Leukocytes
• Lactoferrin
• Occult blood
• Stool cultures (within 2 hours of collection) for Salmonella, Shigella, Campylobacter, E.coli O157:H7.
• O&P
• Flex sig/Colonoscopy
Workup for specific situations:

- Use of antibiotics within previous 2 months - C.Difficile toxin.

- Persistent abdominal pain, fever, mesenteric adenitis, Erythema nodosum - Yersinia enterocolytica, or Y. Pseudotuberculosis.

- Recent shellfish ingestion - Vibrio sp.

- Untreated water/hikers – Giardia, Cryptosporidium
Workup for specific situations:

- HIV/AIDS – Cyclospora, Isospora beli, Cryptosporidium, Microsporidium, Blood cultures or biopsies for MAC, and CMV.
- Immigrant from endemic areas- E. Histolytica.
- Persistent diarrhea with evidence of inflammation raises suspicion for IBD.
Relevant information in Patient’s history:

- Diarrhea 8-10x/d
- Abdominal cramps
- Nausea
- Vomiting
- Imodium/Lomotil
- Ability to drink liquids
- Prev. Constipation?
- ↑Abdominal girth
- Fever – 102.1°F
- Tachycardia 110 (on atenolol)
- Abd exam
- Lab abnormalities
- Neg c-diff x1
- CT scan
Last patient visit:

- Toxic findings: Fever, abdominal distention, tachycardia, no peritoneal signs, leukocytosis, thrombocytosis, electrolyte abnormalities, low albumin.

- CT scan showing Markedly dilated transverse colon of 7.6 cm. Several dilated loops of small bowel without a distinct transition zone. No pneumatosis, free air or free fluid. Mural thickening of the ascending colon near the hepatic flexure.
## Diagnosis of Toxic Megacolon

**Clinical Presentation**
- Diarrhea, bloody diarrhea
- Constipation, Obstipation
- Abdominal pain and tenderness
- Abdominal distention
- Decreased bowel sounds

**Radiographic findings**
- Dilation of transverse or ascending colon >6cm
- Small Bowel and gastric distention
- CT: colonic dilation, diffuse colonic wall thickening, submucosal edema, pericolic stranding, ascites, perforations, abscesses, ascending Pyelophlebitis

**Jalan’s criteria**
- Fever $>101.5 \ (38.6^\circ C)$
- Heart rate $>120$ beats /min
- White blood cell count $>10.5 \ (10^9 /L)$
- Anemia
- Plus one of the following criteria: dehydration, mental changes, electrolyte disturbances, or hypotension.
Toxic Megacolon
Toxic Megacolon

- Toxic Megacolon is a potentially fatal complication of colitis. First recognized as a clinical entity by Marshall et al. in 1950.
Toxic Megacolon

• Definition:
  – Segmental or total colonic distention of >6 cm in the presence of acute colitis and signs of systemic toxicity.
  – Differentiated by other processes that cause colonic distention by its inflammatory trigger and its accompanying toxic manifestations.
Differential diagnosis of Toxic Megacolon

- Ulcerative colitis
- Crohn’s disease
- Salmonella
- Shigella
- Campylobacter
- Entamoeba histolitica
- C. Difficile colitis

- Ischemic colitis
- Immunossuppressed patients, or patients with HIV/AIDS:
  - CMV colitis
  - Cryptosporidia
  - Salmonella
  - Kaposi’s Sarcoma
Risk factors for occurrence

- Cessation or interruption of UC therapy (5 ASA agents or steroids).
- Barium enema and anecdotal reports of colonoscopy as a trigger.
- Drugs that slow colonic motility (narcotic, antidiarrheal or anticholinergics).
- Chemotherapy
- Hypokalemia/ Hypomagnesemia
Workup

- CBC
- Electrolytes
- BUN
- Creatinine
- Albumin
- Blood cultures (Bacteremia in up to 25%)
- Fecal leukocytes
- C.Difficile toxin A and B

- Stool cultures and sensitivity
- Limited colonoscopy (sigmoidoscopy)
  - IBD
  - Pseudomembranous colitis
  - CMV colitis.
Factors associated with a higher mortality (Grenstein et al. 1975)

- Age > 40 years
- Female gender
- Lower albumin level
- Low serum CO2
- High BUN
Management of Toxic Megacolon

- **General:**
  - Intravenous fluids support
  - Correct electrolytes abnormalities
  - Complete bowel rest
  - Discontinue anticholinergics and narcotics (including antidepressants)
  - Rule out Infectious etiology
Management of Toxic Megacolon

- Decompression
  - Rectal tube
  - Nasogastric or long nasointestinal tube
  - Repositioning maneuvers

- Medical care
  - Specific treatment for infections
  - Intravenous corticosteroids for inflammatory Bowel disease
  - Broad spectrum antibiotics
Management of Toxic Megacolon

• Radiology
  – Frequent assessment with plain films
  – Computed tomography scanning may aid in management.

• Absolute indications for Surgical intervention:
  – Failed medical care, progressive dilation
  – Progressive toxicity or dilation
  – Signs of perforation
  – Uncontrollable bleeding
Management of Toxic Megacolon

• Medical therapy for toxic megacolon is directed specifically to the disease process.

• In Particular, Pseudomembranous colitis should be aggressively treated with withdrawal of the offending antibiotics, and either oral or intravenous metronidazole, or oral Vancomycin should be initiated.

• Surgery should not be delayed if clinical parameters continue to worsen.
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Pseudomembranous colitis
Pseudomembranous colitis
(Clostridium difficile colitis)

- Important nosocomial disease which presentation can range from asymptomatic carriage to toxic megacolon with its potential fatal outcomes.

- C. Difficile accounts for 20% of the cases of antibiotic associated diarrhea and for the majority of the cases of colitis associated with antibiotic use.

- Should be suspected in any patient who has received antibiotics within the previous 2 months or whose diarrhea has began 72 hours or more after hospitalization.
Predisposing risk factors

- Antibiotics use for less than 2 months (cefalosporins, ampicillin, clindamycin)

- Recent hospitalization - The endogenous carriage rate is low in the US and Europe (0 to 3%), after admission to a hospital 15 to 21% of the patients become colonized.

- Prolonged hospitalization, ICU stay, abdominal surgery or GI procedures.

- Older age and presence of co-morbidities.

- Diarrhea after 72 hours of hospital admission
Colonization

- Culture of *C. difficile* using selective media to determine carrier rates shows considerable variation depending on the population studied:

  - Healthy adults is 2% to 3%.

  - Patients with antibiotic-associated diarrhea or colitis with positive toxin assays, 90% to 100%.

  - Hospitalized patients without diarrhea, 15% to 30%.

  - Adults who have recently received antimicrobials but do not have diarrhea, 5% to 15%.
Symptoms

- Diarrhea (loose to watery stools). Mucus or occult blood may be present.
- Fever
- Systemic illness
- Abdominal pain
- Abdominal distention
- Toxic megacolon in 0.4 to 3%.
- Bowel perforation can occur in the absence of diarrhea with abdominal pain and distention.
Laboratory

- Hematologic tests:
  - Leukocytosis
  - Hypoalbuminemia
  - Electrolyte disturbances

- Stool Studies:
  - Fecal leukocytes
  - Lactoferrin
  - C.Diff toxin A and B (EIA)
  - Latex agglutination test detects only toxin A
  - Culture for C.Difficile – sensitive, but not specific for toxin producing C.Difficile.
  - Negative culture for other pathogens
Procedures

• Endoscopy:
  – Sigmoidoscopy or colonoscopy is helpful in special situations when the diagnosis is in doubt or the clinical situation demands a rapid diagnosis.
  – Pseudomembranes surrounded by hyperemic mucosa and edema on colonoscopy
Radiology

• CT scans may facilitate the diagnosis but are nonspecific. May show mucosal edema, “thumbprinting”, thickened colon, pancolitis and pericolic inflammation.

• Abdominal radiography may reveal toxic megacolon
Possible diagnoses for this case:

- Inflammatory Bowel disease
  - Ulcerative colitis
  - Crohn’s disease
- Infectious causes
  - C.Difficile colitis (pseudomembranous colitis)
  - Campylobacter Jejuni infection
- Ischemic colitis
Campylobacter Jejuni infection

• Campylobacter jejuni is the most common cause of bacterial gastroenteritis.
  – Approximately 2.4 million cases per year occur in the U.S.

• *Campylobacter* pathogens are small, curved, motile, microaerophilic, gram-negative rods. They also possess a lipopolysaccharide endotoxin.

• Transmission of *C. jejuni* to humans occurs by ingestion of contaminated food or water, including unpasteurized milk and undercooked poultry, or by direct contact with fecal material from infected animals or persons.
Epidemiology

• **Race:** No race predilection exists.

• **Sex:** In people aged 45 years or younger, the *C. jejuni* isolation rate is higher among males than among females. After age 45 years, no sexual predilection exists.

• **Age:** Any age group can be infected with *C. jejuni* enteritis.
Risk Groups

• Persons at increased risk for *Campylobacter* enteritis
  – Occupational exposure to cattle, sheep, and other farm animals
  – Laboratory workers
  – Those in contact with the excreta of infected persons
  – Homosexual men

• Underlying conditions that increase risk for *Campylobacter* bacteremia.
  – Hypogammaglobulinemia, HIV infection, Kwashiorkor
  – Pregnancy, Malignancy, Extremes of age
  – Alcoholism, Diabetes mellitus, Postsplenectomy status
History

• Mild episodes of diarrhea subside within 7 days in 60-70% of cases, last for 2 weeks in 20-30%, and persist longer in 5-10% of cases.

• Inflammatory diarrhea symptoms are indistinguishable from other infectious diarrheas.

• Rarely, in young adults and adolescents, inflammatory diarrhea can be severe and confused with Crohn’s disease and ulcerative colitis.

• Toxic megacolon with massive bleeding may occasionally occur.

• The vast majority of patients recover fully after *C. jejuni* infection within 5 days (range 2-10 d), either spontaneously or after appropriate antimicrobial therapy.
Physical exam

• The abdomen is frequently tender on palpation, especially the right lower quadrant.

• In one third to one half of patients, initial symptoms include periumbilical cramping, intense abdominal pain that mimics appendicitis, malaise, myalgias, headache, and vomiting.

• Fever and bloody stools can also be part of the initial presentation.
Diagnostic studies

• Microbiologic studies:
  – Presumptive diagnosis can be made by darkfield or phase-contrast microscopy.
  – Definitive diagnosis of infection is based on culture.

• Procedures:
  – In patients who undergo proctoscopy secondary to a prolonged course of *Campylobacter* enteritis, normal mucosa is found 50% of the time. Mucosal edema, congestion, friability, and granularity are seen in the remaining half.

• Hematology and blood chemistries
  – Peripheral white blood cell count is usually normal; however, a left shift may occur.
  – Alanine aminotransferase and the erythrocyte sedimentation rate (ESR) may be slightly elevated.
Complications

- Guillain-Barré syndrome.
- Reactive arthritis:
  - Arthritis starts a few days to several weeks after the episode of diarrhea. Joint involvement is usually monoarticular and affects the knees. The course is self-limited, ranging from 1 week to several months.
  - Synovial fluid is sterile.
- Other infrequently reported complications:
  - Reiter syndrome
  - Erythema nodosum
  - Hepatitis
  - Interstitial nephritis
  - Hemolytic-uremic syndrome
  - Immunoglobulin A (IgA) nephropathy
Possible diagnoses for this case:

- Inflammatory Bowel disease
  - Ulcerative colitis
  - Crohn’s disease
- Infectious causes
  - C.Difficile colitis (pseudomembranous colitis)
  - Campylobacter Jejuni infection
- Ischemic colitis
Ischemic colitis
Ischemic colitis

• Disease resulting from the insufficient blood supply to a segment of the colon or its totality causing secondary inflammation.

• It results in various degrees of ischemic necrosis ranging from superficial mucosal necrosis to transmural necrosis.

• Occlusive mesenteric infarction (embolus or thrombosis) has a 90% mortality rate, whereas nonocclusive disease has a 10% mortality rate.
Characteristics

- **Race:** No racial or ethnic predilection for ischemic colitis is reported.

- **Sex:** The male-to-female ratio in ischemic colitis is approximately 1:1.

- **Age:** Ischemic colitis is a disease of the elderly. It is rarely seen in those younger than 60 years. Venous infarction occurs in young patients, usually after abdominal surgery.
Pathophysiology

- Can affect the small bowel alone, colon alone or both.

- Mechanisms:
  - Decreased perfusion due to low cardiac output
  - Occlusive disease of the vascular supply to the bowel.

- Morphologic pattern:
  - (1) transmural infarction,
  - (2) mural infarction when the injury extends from the mucosa into the muscularis.
  - (3) mucosal infarction when ischemic damage is confined to the mucosa.
Causes of mesenteric ischemia

- Hypoperfusion: This may involve heart failure or prolonged shock of any etiology.
- Embolic occlusion
- Atherosclerosis
- Arterial thrombosis
- Venous thrombosis
- Vasculitis
- Thromboangiitis obliterans
- Disseminated intravascular coagulation
- Hypercoagulable states
- Sickle cell disease
- Intra abdominal vascular surgeries.
- Marathon runners
- Translumbar aortography
- Cardiac surgery
- Liver transplantation
- Bowel obstruction,
- Colonic carcinoma
- Trauma
- Drugs.
- Aortic dissection
- Corrosive injury
- Bowel infections, necrotizing enteritis
- Radiation injury
- Arteriovenous fistula between the mesenteric artery and veins.
- Idiopathic
Colonic ischemia

- Due to nonocclusive ischemia.
- Common locations: splenic flexure, descending colon, sigmoid.
- Symptoms: Sudden LLQ pain, Bowel Urgency, red to maroon stools and diarrhea.
- Radiologic findings: thumbprinting of the colonic mucosa last a few days.
- Colonoscopy is the procedure of choice for diagnosis.
Morbidity and Mortality

• Depend on the cause and comorbidities such as underlying cardiac disease, vasculitides, among others.

• The prognosis of colonic ischemia is more favorable than that of other forms of mesenteric ischemia. A transient ischemic episode resolves usually within 1-3 months without sequelae.

• With significant ischemic injury, long strictures may follow. More severe ischemic trauma may cause bowel gangrene and perforation, but this is rare.
Diagnosis

• A reliable diagnosis of ischemic colitis is made by a combination of the history, physical exam, radiologic, endoscopic and histopathologic findings.
Possible diagnoses for this case:

- Inflammatory Bowel disease
  - Ulcerative colitis
  - Crohn’s disease
- Infectious causes
  - C.Difficile colitis (pseudomembranous colitis)
  - Campylobacter Jejuni infection
- Ischemic colitis
Crohn’s disease
Crohn’s disease

- Idiopathic Inflammatory bowel disease, Crohn’s Disease is a condition of chronic inflammation potentially involving any location of the whole GI tract.

- The etiology of Crohn’s disease is largely unknown. Genetic, infectious, immunologic and psychological factors have all been implicated in influencing the development of the disease.
Incidence

- **In the US**: Findings from studies in the United States and Western Europe indicate that the incidence of Crohn disease is 2 cases per 100,000 population. The prevalence is estimated to be 20-40 cases per 100,000 population.
Epidemiology

- **Race:** 2- to 4-fold higher in Jewish populations than in other ethnic groups. Rates are reported to be highest among Caucasians, followed by African Americans and Asians.

- **Sex:** Studies consistently reveal a greater incidence in women than in men 1.2:1.

- **Age:** Crohn’s disease has a bimodal distribution. One early peak occurs in those aged 18-25 years. A smaller peak is observed in those aged 60-80 years.
Characteristics

- The inflammation is often discontinuous but involves all layers from mucosa to serosa, involving mesentery as well as regional lymph nodes.

- Early mucosal involvement consists of longitudinal and transverse aphthous ulcerations, which are responsible for cobblestone appearance. As the disease progress, deep fissures, sinuses and fistulas develop.

- Because of the transmural nature of the disease, mesenteric and perianal manifestations are fairly common.
Risk Factors

• Family History: Crohn’s disease is associated with *HLA-DR1* and *DQw5* genes.

• Smoking: although smokers have a decreased risk for ulcerative colitis, they have an increased risk of Crohn’s disease.

• Oral contraceptives: The risk ratio of developing Crohn’s disease is 2 to 1 in some studies.

• NSAID use increase the risk for CD.
Signs and symptoms

- Fever
- Abdominal pain
- Diarrhea
- Weight loss
- Rectal bleeding is less common.
- Anorectal fistulas, fissures, and perirectal abscess.
- Ileitis - right lower quadrant tenderness with an associated fullness or mass.
- Toxic Megacolon (0 to 20% - 2 to 4%)

Laboratory:
- Anemia
- Leukocytosis
- Elevated ESR
- ASCA (anti saccharomyces cerevisae antibody) may be helpful
Complications

- Intestinal obstruction.

- Fistula formation is common and can cause indolent abscess, malabsorption, cutaneous fistula, persistent urinary tract infection, or pneumaturia.
Extraintestinal manifestations

- Oral aphthous ulcer
- Erythema nodosum
- Osteomalacia
- Anemia
- Osteonecrosis
- Gallstone formation
- Oxalate kidney stones
- Pancreatitis due to therapy
- Amyloidosis
- Thromboembolic complications
- Hepatobiliary disease
- Primary sclerosing cholangitis.
Morbidity and Mortality

• Approximately 15% of the cases of Crohn’s disease appear in those older than 50 years.

• Abscesses develop in approximately 15-20% of patients with Crohn’s disease.

• Obstruction occurs in 20-30% of patients during the course of the disease.

• Fistula formation is a frequent complication of Crohn’s disease of the colon. Complicated fistulas with abscesses or severe underlying bowel disease occur in 50% of patients.

• GI cancer has been the leading cause of mortality in Crohn’s disease.
Possible diagnoses for this case:

- Inflammatory Bowel disease
  - Ulcerative colitis
  - Crohn’s disease
- Infectious causes
  - C.Difficile colitis (pseudomembranous colitis)
  - Campylobacter Jejuni infection
- Ischemic colitis
Ulcerative Colitis
Ulcerative Colitis

• **Background:** Ulcerative colitis is a relatively uncommon, chronic, recurrent inflammatory disease of the colon or rectal mucosa. Often a lifelong illness, the condition has profound emotional and social impact on the affected individual.

• **Pathophysiology:** Ulcerative colitis is defined as continuous idiopathic inflammation of the colonic or rectal mucosa. The rectum is involved in more than 95% of cases.
Incidence

- The annual incidence of ulcerative colitis is 10.4-12 cases per 100,000 people.

- The prevalence rate is 35-100 cases per 100,000 people.
Epidemiology

- **Age:** The incidence of ulcerative colitis peaks in people aged 15-25 years and in people aged 55-65 years, although it can occur in people of any age.

- **Sex:** Ulcerative colitis seems to have a female preponderance. Ulcerative colitis affects 30% more females than males.

- **Race:** Ulcerative colitis occurs more frequently in white people.
Signs and symptoms

- Rectal bleeding
- Diarrhea
- Urgency and tenesmus
- Abdominal cramps, tenderness
- Weight loss
- Mild fever
- Tachycardia
- Dehydration
- Malnutrition
Laboratory

- Anemia
- Thrombocytosis
- Elevated sedimentation rate and C-reactive protein
- Hypoalbuminemia
- Hypokalemia
- Hypomagnesemia
- Elevated alkaline phosphatase: More than 125 U/L suggests primary sclerosing cholangitis
- P ANCA may be helpful
Extracolonic manifestations

- Synovitis
- Ankylosing spondylitis (HLA-B27)
- Sacroiliitis
- Erythema nodosum
- Pyoderma gangrenosum
- Aphthous stomatitis
- Episcleritis
- Iritis
- Primary sclerosing cholangitis
- Uric acid renal stones
- Thromboembolic events.
Risk factors

• Persons with ulcerative colitis are often found to have p-ANCA.

• Family History: Genetic susceptibility (chromosomes 12 and 16) is a factor associated with ulcerative colitis.

• Smoking is negatively associated with ulcerative colitis.

• Appendectomies have a negative association with ulcerative colitis.

• NSAIDs use increase the relapses of UC.
Imaging Studies

• Abdominal radiograph might show colonic dilatation in severe cases, suggesting toxic megacolon. Also, evidence of perforation, obstruction, or ileus can be observed.

• Barium enemas may precipitate toxic megacolon in severe cases. Barium enemas can be performed safely in mild cases.

• CT scan, in general, plays a minor role in the diagnosis of ulcerative colitis. CT scan can show thickening of the colonic wall.
Ulcerative colitis
### Ulcerative colitis vs. Crohn’s

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<thead>
<tr>
<th></th>
<th>Ulcerative Colitis</th>
<th>Crohn’s Disease</th>
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</thead>
<tbody>
<tr>
<td><strong>Distribution</strong></td>
<td>Diffuse inflammation extending from rectum</td>
<td>Rectal sparing, frequent skip lesions</td>
</tr>
<tr>
<td><strong>Inflammation</strong></td>
<td>Diffuse, with mucosal granularity or friability</td>
<td>Focal and asymmetrical, cobblestoning; granularity and friability less common</td>
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<tr>
<td><strong>Ulceration</strong></td>
<td>Small ulcers in a diffusely inflamed mucosa; deep, ragged ulcers in severe disease</td>
<td>Aphthoid ulcers, linear/serpiginous ulceration; intervening mucosa often normal</td>
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<tr>
<td><strong>Colonic lumen</strong></td>
<td>Often narrowed in long-standing chronic disease; strictures very rare</td>
<td>Strictures common</td>
</tr>
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Diagnostic procedures

- Findings on flexible sigmoidoscopy with biopsies can provide the diagnosis of colitis.

- Findings on colonoscopy with biopsy confirm a diagnosis. It is useful for documenting the extent of the disease, for monitoring disease activity, and for surveillance for dysplasia or cancer.
Complications

• **Complications:** Toxic megacolon. Incidence varies 1.6 - 22%.

• The risk of colorectal cancer increases by 0.5-1% per year. Regular surveillance is needed.

• **Prognosis:** Most cases are controlled with medical therapy, with exacerbation on occasion. In more severe cases, surgery results in a cure.
Most Likely Diagnoses

- Inflammatory Bowel disease
- C. difficile colitis

Procedure:
- Limited colonoscopy
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Dr. Pfanner
Response
## Causes and associations with Toxic Megacolon

<table>
<thead>
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<th>Potential triggers and exacerbating factors</th>
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<tr>
<td>Hypokalemia/Hypomagnesemia</td>
</tr>
<tr>
<td>Barium enema</td>
</tr>
<tr>
<td>Discontinuation of steroids</td>
</tr>
<tr>
<td>Narcotics</td>
</tr>
<tr>
<td>Anticholinergics</td>
</tr>
<tr>
<td>Chemotherapy</td>
</tr>
<tr>
<td>Colonoscopy</td>
</tr>
</tbody>
</table>

### Inflammatory
- Ulcerative Colitis
- Crohn’s disease

### Infectious
- Clostridium Difficile
- Salmonella, Shigella, Yersinia, Campylobacter
- Cryptosporidium
- Entamoeba
- CMV

### Ischemia

### Malignancy
- Kaposi’s Sarcoma
Imaging studies – X-Ray:

- The radiologic appearances of ischemic colitis are nonspecific and may be seen in other inflammatory disorders of the colon.

- Dilatation of a part of the colon as well as small bowel, depending on the extent of the disease.

- Mucosal edema (thumbprinting) and pseudopolyp formation (resembling Ulcerative colitis), hoselike appearance of the colon.

- Localized pneumatosis coli may be apparent.
diagnosis

- Crohns Disease
The End

- Proceed to the post test
- Print off the post test
- Complete the post test
- Return the post test to Dr. Sandra Oliver
- TAMUII Rm. 407 i
Post Test Question One

• The most common inflammatory causes of Toxic Megacolon are:
  • 1. ______________________
  • 2. ______________________
Post Test Question 2

Most cases of ulcerative colitis are controlled with
A. Medical therapy
B. Surgical therapy
### Fill in the blanks

<table>
<thead>
<tr>
<th></th>
<th><strong>Ulcerative Colitis</strong></th>
<th><strong>Crohn’s Disease</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Distribution</strong></td>
<td>Diffuse inflammation extending from _______(rectum or splenic flexure)</td>
<td>Rectal sparing, frequent skip lesions</td>
</tr>
<tr>
<td><strong>Inflammation</strong></td>
<td>_______(Diffuse or Focal), with mucosal granularity or friability</td>
<td>Focal and asymmetrical, cobblestoning; granularity and friability less common</td>
</tr>
<tr>
<td><strong>Ulceration</strong></td>
<td>Small ulcers in a diffusely inflamed mucosa; deep, ragged ulcers in severe disease</td>
<td>Aphthoid ulcers, linear/serpiginous ulceration; intervening mucosa often normal</td>
</tr>
<tr>
<td><strong>Colonic lumen</strong></td>
<td>Often narrowed in long-standing chronic disease; strictures very ____ (rare or common)</td>
<td>Strictures_____________ (common or rare)</td>
</tr>
</tbody>
</table>