

Ulcerative Colitis; Crohn Disease

Kulishov S.K.

**prof. of internal medicine No1
department, Poltava State
Medical University, Ukraine**

Plan of lecture

- **Ulcerative Colitis:** etiology, pathogenesis, diagnosis, treatment;
- **Crohn Disease (Regional Enteritis; Granulomatous Ileitis; Granulomatous Ileocolitis) :** etiology, pathogenesis, diagnosis, treatment;

Ulcerative colitis

- Ulcerative colitis is a chronic inflammatory and ulcerative disease arising in the colonic mucosa, characterized most often by bloody diarrhea. Extraintestinal symptoms, particularly arthritis, may occur (Douglas M Heuman, Anastasios A Mihas, Anastasios A Mihas, Jeff Allen Cholelithiasis. MSD publication [1]: <https://www.msdmanuals.com/professional/gastrointestinal-disorders/inflammatory-bowel-disease-ibd/overview-of-inflammatory-bowel-disease>

Etiology and Pathogenesis of Ulcerative colitis [1]

- Patients typically have attacks of bloody diarrhea of varied intensity and duration interspersed with asymptomatic intervals
- Usually an attack begins insidiously, with increased urgency to defecate, mild lower abdominal cramps, and blood and mucus in the stools. Some cases develop after an infection (eg, amebiasis, bacillary dysentery).
- When ulceration is confined to the rectosigmoid, the stool may be normal or hard and dry, but rectal discharges of mucus loaded with red and white blood cells accompany or occur between bowel movements. Systemic symptoms are absent or mild.

Symptoms and Signs [1]

- Ulcerative colitis usually begins in the rectum. It may remain localized to the rectum (ulcerative proctitis) or extend proximally, sometimes involving the entire colon. Rarely, it involves most of the large bowel at once.
- The inflammation caused by ulcerative colitis affects the mucosa and submucosa, and there is a sharp border between normal and affected tissue. Only in severe disease is the muscularis involved. Early in the disease, the mucous membrane is erythematous, finely granular, and friable, with loss of the normal vascular pattern and often with scattered hemorrhagic areas. Large mucosal ulcers with copious purulent exudate characterize severe disease. Islands of relatively normal or hyperplastic inflammatory mucosa (pseudopolyps) project above areas of ulcerated mucosa. Fistulas and abscesses do not occur.

Symptoms and Signs [1]

If ulceration extends proximally, stools become looser and the patient may have > 10 bowel movements per day, often with severe cramps and distressing rectal tenesmus, without respite at night. The stools may be watery or contain mucus and frequently consist almost entirely of blood and pus.

Toxic or fulminant colitis manifests initially with sudden violent diarrhea, fever to 40° C (104° F), abdominal pain, signs of peritonitis (eg, rebound tenderness), and profound toxemia.

Systemic symptoms and signs, more common with extensive ulcerative colitis, include malaise, fever, anemia, anorexia, and weight loss. Extraintestinal manifestations of inflammatory bowel disease (IBD), particularly joint and skin complications, are most common when systemic symptoms are present.

Diagnosis [1]

Diagnosis:

- Stool cultures and microscopy (to exclude infectious causes)
- Sigmoidoscopy with biopsy

Diagnosis of ulcerative colitis is suggested by typical symptoms and signs, particularly when accompanied by extraintestinal manifestations or a history of previous similar attacks.

Diagnosis [1]

- In all patients, stool cultures for enteric pathogens should be done, and *Entamoeba histolytica* should be excluded by examination of fresh stool specimens. When amebiasis is suspected because of epidemiologic or travel history, serologic titers and biopsies should be done. History of prior antibiotic use or recent hospitalization should prompt stool assay for *Clostridioides difficile* (formerly *Clostridium difficile*) toxin.

Diagnosis [1]

- Patients at risk should be tested for HIV, gonorrhea, herpesvirus, chlamydia, and amebiasis. Opportunistic infections (eg, cytomegalovirus, *Mycobacterium avium-intracellulare*) or Kaposi sarcoma must also be considered in immunosuppressed patients. In women using oral contraceptives, contraceptive-induced colitis is possible; it usually resolves spontaneously after hormone therapy is stopped. Stool testing for lactoferrin and fecal calprotectin can be beneficial in differentiating IBD from functional diarrhea.

Lab Test [1]

- Sigmoidoscopy should be done; it allows visual confirmation of colitis and permits direct sampling of stool or mucus for culture and microscopic evaluation, as well as biopsy of affected areas. Although visual inspection and biopsies may be nondiagnostic, because there is much overlap in appearance among different types of colitis, acute, self-limited, infectious colitis can usually be distinguished histologically from chronic idiopathic ulcerative colitis or Crohn colitis. Severe perianal disease, rectal sparing, absence of bleeding, and asymmetric or segmental involvement of the colon indicate Crohn disease rather than ulcerative colitis. Colonoscopy is usually unnecessary initially but should be done electively if inflammation has extended proximal to the reach of the sigmoidoscope.

Lab Test [1]

- *Laboratory tests should be done to screen for anemia, hypoalbuminemia, and electrolyte abnormalities. Liver tests should be done; elevated alkaline phosphatase and gamma-glutamyl transpeptidase levels suggest possible primary sclerosing cholangitis. Perinuclear antineutrophil cytoplasmic antibodies are relatively specific (60 to 70%) for ulcerative colitis. Anti-Saccharomyces cerevisiae antibodies are relatively specific for Crohn disease. However, these tests do not reliably separate the 2 diseases and are not recommended for routine diagnosis. Other possible laboratory abnormalities include leukocytosis, thrombocytosis, and elevated acute-phase reactants (eg, erythrocyte sedimentation rate, C-reactive protein).*

X-rays Investigation [1]

- X-rays are not diagnostic but occasionally show abnormalities. Plain x-rays of the abdomen may show mucosal edema, loss of haustration, and absence of formed stool in the diseased bowel. Barium enema shows similar changes, albeit more clearly, and may also show ulcerations, but the enema should not be done during an acute presentation. A shortened, rigid colon with an atrophic or pseudopolypoid mucosa is often seen after several years of illness. X-ray findings of thumbprinting and segmental distribution are more suggestive of intestinal ischemia or possibly Crohn colitis rather than of ulcerative colitis.

Diagnosis by additional data [1] :

- *Duodenal investigation* – can be conducted only if gallstones are absent! Helps to assess motor function of Chronic cholecystitis
Provides 3 portions of bile for further studying of bile characteristics:
 - o Microscopy – signs of inflammation and lithogenicity of bile;
 - o Culture – determination of bacterial flora;
 - o Biochemical analysis – determination of cholesterol, bile acids, phospholipids in bile.

Treatment [1]

Treatment:

- *Dietary management and loperamide (except in acute severe attacks) for symptom relief*
- *5-Aminosalicylic acid (5-ASA)*
- *Corticosteroids and other drugs depending on symptoms and severity*
- *Antimetabolites, biologic agents, and Janus kinase inhibitors*
- *Sometimes surgery*

Treatment: General management

[1]

- Avoiding raw fruits and vegetables limits trauma to the inflamed colonic mucosa and may lessen symptoms. A milk-free diet may help but need not be continued if no benefit is noted. Loperamide 2 mg orally 2 to 4 times a day is indicated for relatively mild diarrhea; higher oral doses (4 mg in the morning and 2 mg after each bowel movement) may be required for more intense diarrhea. Antidiarrheal drugs must be used with extreme caution in severe cases because they may precipitate toxic dilation. All patients with inflammatory bowel disease should be advised to take appropriate amounts of calcium and vitamin D.
- Routine health maintenance measures (eg, immunizations, cancer screening) should be emphasized.

Treatment of mild left-sided disease

[1]

- Patients with mild to moderate ulcerative proctitis or proctosigmoiditis that does not extend proximally beyond the sigmoid colon are treated with 5-ASA (mesalamine) by enema once a day or twice a day depending on severity. Suppositories are effective for more distal disease and are usually preferred by patients. Corticosteroid and budesonide enemas are slightly less effective but should be used if 5-ASA is unsuccessful or not tolerated. Once remission is achieved, dosage is slowly tapered to maintenance levels. Oral 5-ASA drugs theoretically have some incremental benefit in lessening the probability of proximal spread of disease.

Treatment of moderate or extensive disease **[1]**

- Patients with inflammation proximal to the sigmoid colon or left-sided disease unresponsive to topical agents should receive an oral 5-ASA formulation in addition to 5-ASA enemas. High-dose corticosteroids are added for more severe symptoms; after 1 to 2 weeks, the daily dose is reduced by about 5 to 10 mg each week. Immunomodulator therapy with azathioprine or 6-mercaptopurine can be used in patients who are refractory to maximal doses of 5-ASA and would otherwise need long-term corticosteroid therapy.

Treatment of moderate or extensive disease [1]

- Additionally, infliximab, adalimumab, golimumab, ustekinumab, and vedolizumab are beneficial in some patients and may be considered for those refractory to immunomodulator (thiopurine failure) or corticosteroid therapy as well as those who are corticosteroid-dependent. Moreover, a combination of immunomodulator and anti-TNF therapy is sometimes helpful. Finally, in some patients who fail to respond to corticosteroids, immunosuppressants, or biologics, a trial of the Janus kinase inhibitor, tofacitinib, can be considered.

Treatment of severe disease [1]

- Patients with > 10 bloody bowel movements per day, tachycardia, high fever, or severe abdominal pain require hospitalization to receive high-dose IV corticosteroids. 5-ASA may be continued. IV fluids and blood transfusion are given as needed for dehydration and anemia. The patient must be observed closely for the development of toxic colitis. Parenteral hyperalimentation is sometimes used for nutritional support but is of no value as primary therapy; patients who can tolerate food should eat.

Treatment of severe disease [1]

- Patients who do not respond within 3 to 7 days should be considered for IV cyclosporine or infliximab or else for surgery. Patients who do respond to a corticosteroid regimen are switched within a week or so to prednisone 60 mg orally once a day, which may be gradually reduced at home based on clinical response. Patients who are started on IV cyclosporine and respond to therapy are switched to oral cyclosporine and concomitant azathioprine or 6-mercaptopurine.

Treatment of severe disease [1]

- Oral cyclosporine is continued for about 3 to 4 months, during which time corticosteroids are tapered and cyclosporine levels are closely monitored. Some clinicians recommend prophylaxis against *Pneumocystis jirovecii* pneumonia during the interval of overlapping treatment with corticosteroids, cyclosporine, and an antimetabolite. Tacrolimus, an immunosuppressant also used in transplant patients, seems as effective as cyclosporine and may be considered for use in patients with severe or refractory ulcerative colitis who do not require hospitalization. The trough blood levels should be kept between 10 to 15 ng/mL (12 to 25 nmol/L).

Key Points

- Ulcerative colitis begins in the rectum and may extend proximally in a contiguous fashion without intervening patches of normal bowel.
- Symptoms are intermittent episodes of abdominal cramping and bloody diarrhea.
- Complications include fulminant colitis, which may lead to perforation; long-term, the risk of colon cancer is increased.
- Treat mild to moderate disease with 5-ASA by rectum and, for proximal disease, by mouth.
- Treat extensive disease with high-dose corticosteroids, immunomodulator therapy (eg, azathioprine, 6-mercaptopurine), biologics (eg, infliximab, vedolizumab), or the Janus kinase inhibitor tofacitinib.
- Treat fulminant disease with high-dose IV corticosteroids or cyclosporine and antibiotics (eg, metronidazole, ciprofloxacin) or infliximab; colectomy may be required.
- About one third of patients with extensive ulcerative colitis ultimately require surgery.

Crohn Disease (Regional Enteritis; Granulomatous Ileitis; Granulomatous Ileocolitis)

- Crohn disease is a chronic transmural inflammatory bowel disease that usually affects the distal ileum and colon but may occur in any part of the gastrointestinal tract. Symptoms include diarrhea and abdominal pain. Abscesses, internal and external fistulas, and bowel obstruction may arise. Extraintestinal symptoms, particularly arthritis, may occur. Diagnosis is by colonoscopy and imaging studies. Treatment is with 5-aminosalicylic acid, corticosteroids, immunomodulators, anticytokines, antibiotics, and often surgery. (Walfish A.E.; Rafael Antonio Ching Companioni. MSD publication [2]:
<https://www.merckmanuals.com/professional/gastrointestinal-disorders/inflammatory-bowel-disease-ibd/crohn-disease>)

Etiology and Pathogenesis of Crohn Disease [2]

- Crohn disease begins with crypt inflammation and abscesses, which progress to tiny focal aphthoid ulcers. These mucosal lesions may develop into deep longitudinal and transverse ulcers with intervening mucosal edema, creating a characteristic cobblestoned appearance to the bowel. Transmural spread of inflammation leads to lymphedema and thickening of the bowel wall and mesentery. Mesenteric fat typically extends onto the serosal surface of the bowel.

Etiology and Pathogenesis of Crohn Disease [2]

Mesenteric lymph nodes often enlarge. Extensive inflammation may result in hypertrophy of the muscularis mucosae, fibrosis, and stricture formation, which can lead to bowel obstruction.

Abscesses are common, and fistulas often penetrate into adjoining structures, including other loops of bowel, the bladder, or psoas muscle. Fistulas may even extend to the skin of the anterior abdomen or flanks. Independently of intra-abdominal disease activity, perianal fistulas and abscesses occur in 25 to 33% of cases; these complications are frequently the most troublesome aspects of Crohn disease.

Noncaseating granulomas can occur in lymph nodes, peritoneum, the liver, and all layers of the bowel wall. Although pathognomonic when present, granulomas are not detected in about half of patients with Crohn disease. The presence of granulomas does not seem to be related to the clinical course.

Etiology and Pathogenesis of Crohn Disease [2]

- Segments of diseased bowel are sharply demarcated from adjacent normal bowel (called skip areas), hence the name regional enteritis.
- • About 35% of Crohn disease cases involve the ileum alone (ileitis).
- • About 45% involve the ileum and colon (ileocolitis), with a predilection for the right side of the colon.
- • About 20% involve the colon alone (granulomatous colitis), most of which, unlike ulcerative colitis, spares the rectum.
- The ileum is inflamed in about 80% of cases of Crohn disease.
- Occasionally, the entire small bowel is involved (jejunoileitis). The stomach, duodenum, or esophagus is clinically involved only rarely, although microscopic evidence of disease is often detectable in the gastric antrum, especially in younger patients. In the absence of surgical intervention, the disease almost never extends into areas of small bowel that are not involved at first diagnosis.

Crohn Disease: Classification, Complications

[2]

Classification

- Crohn disease is categorized into 3 principal patterns: (1) primarily inflammatory, which after several years commonly evolves into (2) primarily stenotic or obstructing or (3) primarily penetrating or fistulizing.
- These different clinical patterns dictate different therapeutic approaches. Some genetic studies suggest a molecular basis for this classification.

Complications

- There is an increased risk of cancer in affected small-bowel segments. Patients with colonic involvement have a long-term risk of colorectal cancer equal to that of ulcerative colitis, given the same extent and duration of disease. Chronic malabsorption may cause nutritional deficiencies, particularly of vitamins D and B12.
- Toxic megacolon is a rare complication of colonic Crohn disease. It is a clinical syndrome of ileus accompanied by radiographic evidence of colonic dilation; many cases must be treated aggressively with surgical intervention.

Symptoms and Signs [2]

- The most common initial manifestations of Crohn disease are
 - Chronic diarrhea with abdominal pain, fever, anorexia, and weight loss
 - The abdomen is tender, and a mass or fullness may be palpable.
- Gross rectal bleeding is unusual except in isolated colonic disease, which may manifest similarly to ulcerative colitis. Some patients present with an acute abdomen that simulates acute appendicitis or intestinal obstruction. About 33% of patients have perianal disease (especially fissures and fistulas), which is sometimes the most prominent or even initial complaint.

Symptoms and Signs [2]

- With recurrent disease, symptoms vary. Pain is most common and occurs with both simple recurrence and abscess formation. Patients with severe flare-up or abscess are likely to have marked tenderness, guarding, rebound, and a general toxic appearance. Stenotic segments may cause bowel obstruction, with colicky pain, distention, obstipation, and vomiting. Adhesions from previous surgery may also cause bowel obstruction, which begins rapidly, without the prodrome of fever, pain, and malaise typical of obstruction due to a Crohn disease flare-up.

Symptoms and Signs [2]

- An enterovesical fistula may produce air bubbles in the urine (pneumaturia). Draining cutaneous fistulas may occur. Free perforation into the peritoneal cavity is unusual.
- Chronic disease causes a variety of systemic symptoms, including fever, weight loss, malnutrition, and other extraintestinal manifestations of IBD.

Diagnosis [2]

Diagnosis

- Barium x-rays of the small bowel
- Abdominal CT (conventional or CT enterography)
- Sometimes barium enema, magnetic resonance (MR) enterography, upper endoscopy, colonoscopy, and/or video capsule endoscopy

Crohn disease should be suspected in a patient with inflammatory or obstructive symptoms or in a patient without prominent GI symptoms but with perianal fistulas or abscesses or with otherwise unexplained arthritis, erythema nodosum, fever, anemia. A family history of Crohn disease also increases the index of suspicion.

Similar symptoms and signs (eg, abdominal pain, diarrhea) may be caused by other GI disorders, particularly ulcerative colitis. Differentiation from ulcerative colitis may be an issue in the 20% of cases in which Crohn disease is confined to the colon. However, because treatment is similar, this distinction is critical only when surgery or experimental therapy is contemplated.

Diagnosis [2]

- Patients presenting with an acute abdomen (either initially or during a relapse) should have flat and upright abdominal x-rays and an abdominal CT scan. These studies may show obstruction, abscesses or fistulas, and other possible causes of an acute abdomen (eg, appendicitis). Ultrasonography may better delineate gynecologic pathology in women with lower abdominal and pelvic pain.
- If initial presentation is less acute, an upper GI series with small-bowel follow-through and spot films of the terminal ileum is preferred over conventional CT. However, newer techniques of CT or MR enterography, which combine high-resolution CT or MR imaging with large volumes of ingested contrast, are becoming the procedures of choice in some centers.

Diagnosis [2]

- These imaging studies are virtually diagnostic if they show characteristic strictures or fistulas with accompanying separation of bowel loops.
- If findings are questionable, CT enteroclysis or video capsule enteroscopy may show superficial aphthous and linear ulcers. Barium enema x-ray may be used if symptoms seem predominantly colonic (eg, diarrhea) and may show reflux of barium into the terminal ileum with irregularity, nodularity, stiffness, wall thickening, and a narrowed lumen. Differential diagnoses in patients with similar x-ray findings include cancer of the cecum, ileal carcinoid, lymphoma, systemic vasculitis, radiation enteritis, ileocecal tuberculosis, and ameboma.

Diagnosis [2]

- In atypical cases (eg, predominantly diarrhea, with minimal pain), evaluation is similar to suspected ulcerative colitis, with colonoscopy (including biopsy, sampling for enteric pathogens, and, when possible, visualization of the terminal ileum). Upper GI endoscopy may identify subtle gastroduodenal involvement even in the absence of upper GI symptoms.

Additional Investigation [2]

- Laboratory tests should be done to screen for anemia, hypoalbuminemia, and electrolyte abnormalities. Liver tests should be done; elevated alkaline phosphatase and gamma-glutamyl transpeptidase levels in patients with major colonic involvement suggest possible primary sclerosing cholangitis. Leukocytosis or increased levels of acute-phase reactants (eg, erythrocyte sedimentation rate, C-reactive protein) are nonspecific but may be used serially to monitor disease activity.
- To detect nutritional deficiencies, levels of vitamin D and B12 should be checked every 1 to 2 years. Additional laboratory measurements, such as levels of water-soluble vitamins (folic acid and niacin), fat-soluble vitamins (A, D, E and K), and minerals (zinc, selenium, and copper), may be checked when deficiencies are suspected.

Additional Investigation [2]

- All patients with inflammatory bowel disease (IBD), whether male or female, young or old, should have their bone mineral density monitored, usually by dual-energy x-ray absorptiometry (DXA) scan.
- Perinuclear antineutrophil cytoplasmic antibodies are present in 60 to 70% of patients with ulcerative colitis and in only 5 to 20% of patients with Crohn disease. Anti-*Saccharomyces cerevisiae* antibodies are relatively specific for Crohn disease. However, these tests do not reliably separate the 2 diseases and they are not recommended for routine diagnosis. Additional antibodies such as anti-OmpC and anti-CBir1 are now available, but the clinical value of these supplementary tests is uncertain; some studies suggest that high titers of these antibodies have adverse prognostic implications.

Treatment [2]

Treatment

- Loperamide or antispasmodics for symptom relief
- 5-Aminosalicylic acid (5-ASA) or antibiotics
- Other drugs depending on symptoms and severity (eg, immunomodulating drugs and biologic agents)
- Sometimes surgery

Treatment [2]

- General management:
- Cramps and diarrhea may be relieved by oral administration of loperamide 2 to 4 mg or antispasmodic drugs up to 4 times a day (ideally before meals). Such symptomatic treatment is safe, except in cases of severe, acute Crohn colitis, which may progress to toxic colitis as in ulcerative colitis. Hydrophilic mucilloids (eg, methylcellulose or psyllium preparations) sometimes help prevent anal irritation by increasing stool firmness. Dietary roughage is to be avoided in stricturing disease or active colonic inflammation.

Treatment [2]

- Mild to moderate disease
- This category includes ambulatory patients who tolerate oral intake and have no signs of toxicity, tenderness, mass, or obstruction. 5-ASA (mesalamine) is commonly used as first-line treatment. Pentasa® is favored for small-bowel disease, and Asacol® HD is favored for distal ileal and colonic disease. However, the benefits of any 5-ASA drug for small-bowel Crohn disease are modest, and many experts advocate not using it in small-bowel Crohn disease.
- Antibiotics are considered a first-line agent by some clinicians, or they may be reserved for patients not responding to 4 weeks of 5-ASA; their use is strictly empiric. With any of these drugs, 8 to 16 weeks of treatment may be required.
- Responders should receive maintenance therapy.

Treatment [2]

Moderate to severe disease

- Patients without fistulas or abscesses but with significant pain, tenderness, fever, or vomiting, or those who have not responded to treatment for mild disease, often have rapid relief of symptoms when given corticosteroids, either oral or parenteral. Oral prednisone or prednisolone may act more rapidly and reliably than oral budesonide, but budesonide has somewhat fewer adverse effects and is considered the corticosteroid of choice in many centers, especially in Europe.

Treatment [2]

- Patients who do not respond rapidly to corticosteroids, or those whose doses cannot be tapered within a few weeks, must not be maintained on these drugs and require different therapy.
- An antimetabolite (azathioprine, 6-mercaptopurine, or methotrexate), an anti-tumor necrosis factor (TNF) agent (infliximab, adalimumab, or certolizumab pegol), or a combination of both, can be used as 2nd-line therapy after corticosteroids, and even as first-line therapy in preference to corticosteroids. These drugs, guided by measurements of drug and antibody levels, achieve clinical success in most cases.

Treatment [2]

- When these lines of treatment fail in patients for whom surgery is not feasible or appropriate, newer biologic drugs including anti-integrins (eg, vedolizumab) or an anti-IL-12/23 antibody (eg, ustekinumab) can be used. Furthermore, other biologic agents are emerging rapidly.
- Obstruction is managed initially with nasogastric suction and IV fluids. Obstruction due to uncomplicated Crohn disease should resolve within a few days and therefore does not require either specific anti-inflammatory therapy or parenteral nutrition; absence of prompt response, however, indicates a complication or another etiology and requires immediate surgery.

Treatment [2]

- Fulminant disease or abscess
- Patients with toxic appearance, high fever, persistent vomiting, rebound, or a tender or palpable mass must be hospitalized for administration of IV fluids and antibiotics. Abscesses must be drained, either percutaneously or surgically. IV corticosteroids or biologic agents should be given only when infection has been ruled out or controlled. If there is no response to corticosteroids and antibiotics within 5 to 7 days, surgery is usually indicated.

Treatment [2]

- **Fistulas**
- Perianal fistulas are treated initially with metronidazole and ciprofloxacin. Patients who do not respond in 3 to 4 weeks may receive an immunomodulator (eg, azathioprine, 6-mercaptopurine), with or without an induction regimen of infliximab or adalimumab for more rapid response. Anti-TNF therapy (infliximab or adalimumab) can also be used alone. Cyclosporine or tacrolimus is an alternative, but fistulas often relapse after treatment.
- Endoscopic ultrasound-guided placement of fibrin glue, or use of a seton drain (a piece of suture material temporarily left in the fistula to allow it to drain), may help some patients with more complex or refractory perianal fistulas. Severe refractory perianal fistulas may require temporary diverting colostomy but almost invariably recur after reconnection; hence, diversion is more appropriately considered a preparation for definitive surgery or at best an adjunct to infliximab or adalimumab rather than a primary treatment.

Treatment [2]

- **Maintenance therapy**
- Patients who require only 5-ASA or an antibiotic to achieve remission of Crohn disease can be maintained on that drug. Patients requiring acute treatment with corticosteroids or anti-TNF agents typically require azathioprine, 6-mercaptopurine, methotrexate, anti-TNF therapy, or combination therapy for maintenance. Many if not most patients brought into remission with an anti-TNF agent will require escalation of the dose or shortening of the treatment intervals within a year or two. Systemically active corticosteroids are neither safe nor effective for long-term maintenance, although budesonide has been shown to delay relapse with fewer adverse effects. Patients who respond to anti-TNF therapy for acute disease but who are not well maintained on antimetabolites may stay in remission with repeat doses of anti-TNF agents.
- Monitoring during remission can be done by following symptoms and doing blood tests and does not require routine x-rays or colonoscopy (other than regular surveillance for dysplasia after 7 to 8 years of disease).

Surgery [2]

Even though about 70% of patients ultimately require an operation, surgery for Crohn disease is often done reluctantly. It is best reserved for recurrent intestinal obstruction or intractable fistulas or abscesses. Resection of the involved bowel may ameliorate symptoms but does not cure the disease, which is likely to recur even after resection of all clinically apparent lesions.

The recurrence rate, defined by endoscopic lesions at the anastomotic site, is

- > 70% at 1 year
- > 85% at 3 years

Defined by clinical symptoms, the recurrence rate is about

- 25 to 30% at 3 years
- 40 to 50% at 5 years

Surgery [2]

- Ultimately, further surgery is required in nearly 50% of cases. However, recurrence rates seem to be reduced by early postoperative prophylaxis with 6-mercaptopurine or azathioprine, metronidazole, or infliximab. Moreover, when surgery is done for appropriate indications, almost all patients have improved quality of life.
- Because smoking increases the risk of recurrence, especially in women, smoking cessation should be encouraged.

Key Points [2]

Key Points:

- Crohn disease typically affects the ileum and/or colon but spares the rectum (which is invariably affected in ulcerative colitis).
- Intermittent areas of diseased bowel are sharply demarcated from adjacent normal bowel (called skip areas).
- Symptoms primarily involve episodic diarrhea and abdominal pain; gastrointestinal bleeding is rare.
- Complications include abdominal abscesses and enterocutaneous fistulas.
- Treat mild to moderate disease with 5-aminosalicylic acid and/or antibiotics (eg, metronidazole, ciprofloxacin, rifaximin).
- Treat severe disease with corticosteroids and sometimes immunomodulators (eg, azathioprine) or biologics (eg, infliximab, vedolizumab, ustekinumab).
- About 70% of patients ultimately require an operation, typically for recurrent intestinal obstruction, intractable fistulas, or abscesses.

Ulcerative colitis as visualized with a colonoscope ([3] Marc D Basson,
Chief Editor BS Anand, medscape publication - "Ulcerative Colitis"
<https://emedicine.medscape.com/article/183084-overview>)



Colonoscopic image of a large ulcer and inflammation of the descending colon in a 12-year-old boy with Crohn disease ([4] Leyla J Ghazi, Specialty Editor Board BS Anand, Chief Editor Praveen K Roy. Medscape publication "Crohn disease" <https://emedicine.medscape.com/article/172940-overview>)

