Ulcerative Colitis: Introduction

Inflammatory bowel disease encompasses two idiopathic, chronic, inflammatory diseases: Crohn's disease and ulcerative colitis. Crohn's disease and ulcerative colitis are disorders of unknown cause involving genetic and immunological influence on the gastrointestinal tract's ability to distinguish foreign from self-antigens. They share many overlapping epidemiological, clinical and therapeutic characteristics. In some patients, it is not possible to distinguish which form of inflammatory bowel disease is present (Figure 2).

There are, however, important pathological and clinical differences that distinguish these inflammatory disease processes. Clinically, Crohn's disease tends to present more frequently with abdominal pain and perianal disease, whereas ulcerative colitis is more often characterized by gastrointestinal bleeding.

Cobblestoning mucosa and aphthous or linear ulcers characterize the endoscopic appearance of Crohn's disease. Ulcerative colitis presents with diffuse continuous involvement of the mucosa. Radiographic studies of patients with Crohn's disease characteristically show fistulas, asymmetry and ileal involvement. In contrast, radiographic studies of patients with ulcerative colitis show continuous disease without fistulizing or ileal disease (Figure 3).

Pathologically, Crohn's disease features mucosal discontinuity, transmural involvement and granulomas. In contrast, ulcerative colitis does not. Crypt abscesses and granulomas are present only in Crohn's disease. Figure 4 compares the appearance of the colon, the histology, and endoscopic views of normal, Crohn's disease, and ulcerative colitis patients.

Ulcerative colitis (UC) is an idiopathic inflammatory bowel disease that occurs more often in industrialized countries. This disease affects both men and women similarly. The disease may be acute and chronic with unpredictable relapses and remissions. Major advances have been made in many aspects of inflammatory bowel disease.
bowel disease, including new information on the molecular basis of the disease, epidemiological considerations, immunology and genetics. The clinical and scientific understanding of ulcerative colitis has been greatly expanded far beyond our earlier knowledge.

What is Ulcerative Colitis?

Ulcerative colitis is an idiopathic inflammatory bowel disease that affects the colonic mucosa and is clinically characterized by diarrhea, abdominal pain and hematochezia. The extent of disease is variable and may involve only the rectum (ulcerative proctitis), the left side of the colon to the splenic flexure, or the entire colon (pancolitis). The severity of the disease may also be quite variable histologically, ranging from minimal to florid ulceration and dysplasia. Carcinoma may develop. The typical histological (microscopic) lesion of ulcerative colitis is the crypt abscess, in which the epithelium of the crypt breaks down and the lumen fills with polymorphonuclear cells. The lamina propria is infiltrated with leukocytes. As the crypts are destroyed, normal mucosal architecture is lost and resultant scarring shortens and can narrow the colon.

Systemic and Extra-Colonic Manifestations

Arthritic complications may occur in as many as 26% of patients with ulcerative colitis. Spondylitis occurs in 3% of these patients. The arthritic symptoms may appear before the inflammatory bowel disease and do not necessarily follow the course of the intestinal disease. Twelve to 23% of patients with ulcerative colitis have peripheral arthritis, which affects large, weight-bearing joints such as knees or ankles. Arthritis signs and symptoms usually accompany exacerbations of ulcerative colitis.

Nineteen percent of patients with ulcerative pancolitis experience dermatological changes. Erythema nodosum and pyoderma gangrenosum are commonly associated with this disease. Other dermatological sequelae include dermatitis, erythematous rash, psoriasis, carcinoma, urticaria, pityriasis, lupus erythematosus, vitiligo and ecchymosis.

Ocular manifestations of ulcerative colitis occur in 5% of patients with extensive disease or with Crohn's disease, and may include anterior uveitis, episcleritis and keratoconjunctivitis. Symptoms of these complications include headache, photophobia, blurred vision, burning and increased secretions from the eyes (Figure 6).

In most situations, extraintestinal manifestations respond to standard medical therapy. On rare occasions, a total proctocolectomy may be necessary to control severe extraintestinal manifestations of this disease.
Classification

The extent of colonic mucosal involvement and severity of disease correlate with the clinical manifestations of ulcerative colitis. Approximately one-third of all patients with ulcerative colitis have involvement limited to the rectum (the distal 15 cm of the large intestine) or ulcerative proctitis. Ulcerative proctitis is endoscopically characterized by edema, erythema and loss of vascular markings. Granularity, friability, and frank ulceration are also seen in more severe disease.

Figure 7. Extent of bowel involvement in different degrees of ulcerative colitis.

Distal or left-sided colitis is found in patients in whom the inflammatory process extends from the rectum 40 cm. Disease activity does not extend beyond the splenic flexure, and there is evidence of chronic inflammation and chronic architectural distortion.

Pancolitis involves the portion of the colon beyond splenic flexure. It is characterized by hematochezia and diarrhea, and may be accompanied by abdominal pain and cramps, fever, and/or weight loss with persistent inflammation. Normal haustral markings disappear with generalized shortening and tubularization of the colon. In severe disease, the mucosa may be described as nodular with pseudopolyps, a reticular pattern, and discrete ulcer craters.

Incidence

The incidence of ulcerative colitis has remained fairly constant in those areas for which data are available for a number of years. Ulcerative colitis has been reported between 1.0 and 15.0 cases per 100,000. In general, the rates are highest in the Scandinavian countries, Great Britain and North America. The disease is uncommon in Asia, Africa and South America, although good data are generally lacking from underdeveloped countries where the rates seem to be low. Prevalence is higher among Jewish people born in Europe and the United States (Ashkenazi Jews) than among those born in Asia and Africa. The literature reports a slightly higher incidence of ulcerative colitis in females than males. It is most likely to occur in early adulthood, but disease presentation can occur in the fifth or sixth decade, and occasionally in the seventh or eighth decade. Diet, breast-feeding, oral contraceptives, and the cessation of cigarette smoking have been implicated as risk factors for ulcerative colitis. Studies indicate a decreased risk of ulcerative colitis for current smokers, however, former smokers are at increased risk of developing the disease.

Symptoms

The predominant symptom in ulcerative colitis is diarrhea, which can be associated with frank blood in the stool. The patient has frequent bowel movements, which may be small in volume, as a result of irritability of the inflamed rectum (proctitis). Other symptoms include abdominal or rectal pain, fever and weight loss. Although diarrhea is the dominant complaint in patients with ulcerative colitis, some patients may complain of constipation and rectal spasm.
Ulcerative Colitis: Anatomy

The lower gastrointestinal tract may be divided into the cecum, the ascending colon, the transverse colon, the descending colon, the sigmoid colon and the rectum. The large intestine (colorectum) begins at the cecum, which is a pouch approximately 2–3 inches long. Ileal contents empty into the cecum through the ileocecal valve. The appendix extends from the base of the cecum. The ascending colon rises from the cecum along the right posterior wall of the abdomen, under the ribs to the undersurface of the liver. At this point it turns toward the midline (hepatic flexure), becoming the transverse colon. The transverse portion crosses the abdominal cavity toward the spleen, goes high up into the chest under the ribs, and turns downward at the splenic flexure. Continuing along the left side of the abdominal wall to the rim of the pelvis, the descending colon turns medially and inferiorly to form the S-shaped sigmoid (sigma-like) colon. The rectum extends from the sigmoid colon to the pelvic floor muscles, where it continues as the anal canal terminating at the anus (Figure 8). The anal canal is approximately 4 cm long.

The large intestine is approximately 5–6 feet long and 2½ inches in diameter. It is the site of salt and water absorption. Glands secrete large quantities of alkaline mucus that lubricate the intestinal contents and neutralize acids formed by bacteria in the intestine. These bacteria aid in decomposition of undigested food residue, unabsorbed carbohydrates, amino acids, cell debris, and dead bacteria through the process of segmentation and putrefaction. Short-chain fatty acids, formed by bacteria from unabsorbed complex carbohydrates, provide an energy source for the cells of the left colon. Maintenance of potassium balance is also assigned to the colon, where the epithelium absorbs and secretes potassium (K) and bicarbonate.
Ulcerative Colitis: Causes

Genetics
Inheritance on a polygenetic basis seems to play a role in the etiology of ulcerative colitis in about 12–15% of cases. The most firmly established and quantitatively greatest risk factor for developing ulcerative colitis is a family history. The factors responsible for variable expression of this heritable susceptibility are not known. Also, the fact that migrants to developed countries appear to develop higher rates of disease, and the rates among Jews vary by country, support an important environmental component to risk as well. Evidence of higher rates of ulcerative colitis in urban areas raises the issue of a transmissible agent that may be responsible for disease expression or increased susceptibility.

Environmental
Environmental factors that may potentiate the onset of ulcerative colitis are currently under investigation. Such risk factors include diet, breast-feeding and other perinatal events, occupation and social class, oral contraceptive use, and, most impressively, the cessation of cigarette smoking. Although the "protective" factor in tobacco smoke is unknown, several preliminary trials have shown promising results.

Pathogenesis
The pathogenesis of ulcerative colitis remains unknown. Several theories have been proposed that implicate vascular impairment, autoimmune mechanisms, bacterial-immunological interactions, and allergic or hypersensitivity reactions.

Recent literature on inflammatory bowel disease (IBD), Crohn's disease, and ulcerative colitis reports an intensive search for the antigens that trigger the immune response in inflammatory bowel disease. There are three major hypotheses as to these antigenic triggers. One hypothesis is that these triggers are microbial pathogens, as yet unidentified. According to this theory, the immune response in IBD is an appropriate but ineffective response to these pathogens. The second hypothesis as to the antigenic trigger in IBD is that there is some common dietary antigen or nonpathogenic microbial agent to which the patient mounts an abnormal immune response. It has been hypothesized that patients with IBD are genetically programmed to mount an intense immune response to some common luminal antigen (dietary or microbial) to which most people do not respond.

Diet is a major source of antigens in the intestinal lumen. Dietary antigens are capable of triggering immune responses. One of the foods implicated in the pathogenesis of IBD is cow's milk. Patients with IBD and Crohn's disease demonstrate an increased incidence of antibodies to cow's milk protein. In patients with IBD, cow's milk proteins and other dietary antigens have abnormal access to the lamina propria because of the defect in the epithelial cell monolayer caused by inflammation. Normally, the intestinal epithelium is a barrier between the immune cells of the lamina propria and luminal antigens; however, in IBD, the immune cells of the lamina propria are exposed to numerous luminal antigens. These luminal antigens are capable of triggering immune responses. As a result, specific immune responses to the etiological agent may be overwhelmed by immune responses to thousands of luminal antigens that pass through the damaged epithelium.

The third hypothesis relating to antigenic triggers postulates that an antigen is expressed on the patient's own cells, particularly on intestinal epithelial cells. Theoretically, the patient mounts an appropriate immune response against some luminal antigen; but because of similarities between proteins on the epithelial cells and the lumen antigen, the patient's immune system also attacks the epithelial cells. Under this autoimmune theory, the immune response is directed toward the epithelial cells, and the cells are destroyed by one of two immune effector mechanisms—either antibody-dependent cellular cytotoxicity or direct cell-mediated cytotoxicity (Figure 9).
Ulcerative Colitis: Diagnosis

Overview

Evaluations at initial presentation, at the beginning of each subsequent attack, and at multiple points during each attack are required to assess the clinical picture. The extent of the evaluation should be guided by the presentation. The milder the presentation, the less extensive or invasive the evaluation.

The frequency and severity of diarrhea is a good indicator of the severity of disease. Six or more bowel movements per day are associated with severe disease. The increase in frequency of bowel movements during an attack, as compared to the normal number of bowel movements, is more informative than the absolute number. Fever, hypotension, and tachycardia are markers for the presence of severe disease and necessitate more extensive evaluation in a hospital setting. Nocturnal bowel movements are also crucial in the history to determine severity. The differential diagnosis in ulcerative colitis includes other forms of inflammatory bowel disease, including Crohn’s disease, diverticular inflammation and hemorrhage, collagenous colitis, ischemic bowel disease, radiation colitis, and infectious etiologies including the following organisms: Campylobacter, Shigella, Clostridium difficile, amebiasis, and Escherichia coli 0157:H7.

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<th>Non-Invasive Diagnostic Tests</th>
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<td>Levels of hemoglobin, leukocyte count, and erythrocyte sedimentation rate reflect disease activity. Hypoalbuninemia and electrolyte disorders, such as hyperkalemia, are often seen with severe diarrhea. These studies play a role in clinical evaluation and are useful in confirming the initial impression and in following the subsequent clinical course of remission and exacerbations.</td>
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<th>Non-Invasive Diagnostic Imaging</th>
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<td>Plain abdominal x-rays demonstrate the gaseous outline of the transverse colon in the acutely ill patient. Shortening of the colon and loss of haustral markings can also be demonstrated by plain films, as well as a double-contrast barium enema. Indications of ulcerative disease include loss of mucosal detail, cobblestone filling defects, and segmental areas of involvement. Contrast studies are a sensitive radiological diagnostic tool to determine the extent of ulcerative colitis. Currently, the most common radiological procedures include the small-bowel series, enteroclysis, barium enema and upper gastrointestinal films.</td>
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<th>Small-Bowel Series</th>
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<td>This is a fast, safe procedure for visualization of the small bowel. The patient drinks a barium suspension and overhead abdominal radiographs are taken at 20–30 minute intervals. When the barium reaches the right colon, fluoroscopy is performed while moving the patient in various positions to unwind superimposed bowel loops. Compression spot radiographs are obtained with attention to the terminal ileum.</td>
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Enteroclysis
Enteroclysis is more sensitive for focal lesions (such as adhesions), but has a higher rate of complications and technical difficulty. With the patient mildly sedated, a tube is passed through the nose and advanced into the jejunum. Under constant fluoroscopic imaging, barium is infused through the tube with a methylcellulose solution, resulting in distension and coating of small-bowel loops. The appearance is similar to a double-contrast enema.

**Barium Enema**

This is a safe, effective tool for evaluation of patients with ulcerative colitis. It demonstrates ulcer depth and fistulas. A high-density barium preparation is administered through a rectal tube. Under fluoroscopy, air is introduced until the entire colon is distended and coated with barium. Spot films are taken during the filling of the colon and a series of overhead films are taken after the patient has been positioned to demonstrate the whole colon. Post-evacuation films are also obtained (Figure 10).

**Upper Gastrointestinal Films**

These films allow evaluation of the esophagus, stomach and duodenum. Examination can be performed using single- or double-contrast techniques. In the single-contrast study, the patient drinks a barium suspension. Fluoroscopic spot radiographs are taken. During the double-contrast examination, the patient ingests effervescent gas crystals followed by a barium solution. Air distends the upper gastrointestinal tract, which is coated with barium, and a series of spot radiographs are obtained.

**Radiological Diagnosis**

**Computed Tomography (CT)**

CT scanning is a valuable tool in the diagnostic evaluation of patients with ulcerative colitis and is complementary to contrast exams. CT can accurately image the bowel wall and the extraluminal disease extension. Oral contrast and/or IV contrast is administered to the patient before the examination, allowing for opacification of the stomach, small bowel and colon (Figure 11).

**Magnetic Resonance Imaging (MRI)**

MRI is an ideal imaging tool, but its application is limited. Technical advances have reduced the imaging time and decreased motion artifacts. The technique has demonstrated usefulness in evaluating the severity of disease and colonic wall thickness.

**Endoscopic Diagnosis**

Endoscopy is essential at initial presentation to establish diagnosis and determine the extent of disease. It may also be useful at the time of subsequent attacks to determine recurrence of ulcerative colitis or extension of disease activity, and for surveillance for dysplasia.

**Flexible Sigmoidoscopy**

Lower abdominal symptoms should be evaluated by flexible sigmoidoscopy. This allows examination from the rectum through the sigmoid colon and takes approximately 10–20 minutes (Figure 12).
This procedure is simple to perform and easily tolerated. Patients may experience slight cramping or pressure in the lower abdomen; however, as soon as air leaves the colon the discomfort resolves. This examination allows for a limited endoscopic view when the patient is known to have only limited ulcerative proctitis.

Colonoscopy
Colonoscopy is a procedure that takes 30–60 minutes and allows examination of the entire large intestine from the rectum through the colon to the terminal ileum. Sedation is administered so the patient does not experience significant discomfort. The colon must be completely empty for colonoscopic examination to be thorough and safe. Patients are routinely placed on a liquid diet for 1–2 days before the examination and administered oral laxative and/or enemas to clear the colon. The physician inserts a long, flexible, lighted colonoscope into the rectum and guides it into the colon and potentially to the terminal ileum (Figure 14).

The colonoscope transmits images of the inside of the colon to a monitor, viewable by the physician. Air may be insufflated into the colon to improve visibility. During the procedure, a variety of instruments can be utilized through the biopsy channel of the scope (snare or forceps for obtaining tissue specimens) (Figure 15). Colonoscopy is a sensitive and specific diagnostic tool in ulcerative colitis.
Figure 15. Biopsy of colonic mucosa.
Ulcerative Colitis: Therapy

Overview
The primary goal of therapy in ulcerative colitis is to reduce acute and chronic inflammation ultimately resulting in complete clinical and endoscopic remission. Medical therapies, as well as surgical intervention, are the current modalities for treatment of ulcerative colitis. Approximately 70% of patients respond favorably to medical regimens and go into remission. Surgery cures ulcerative colitis. Surgery is indicated for those patients who are unresponsive to medical therapy and have a severely compromised quality of life. Growth failure in children, life-threatening complications such as severe bleeding, toxic megacolon, impending perforation, intolerance to immunosuppression, colonic strictures, and dysplasia or carcinoma are also indications for surgery.

Medical Therapy
Anti-inflammatory drugs (adrenocorticosteroids and compounds containing 5-aminosalicylic acid) are the mainstays of medical therapy. These medications in a variety of forms are used orally and topically to reduce inflammation of the colon and rectum.

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<th>Drug</th>
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<th>Response rate</th>
<th>Initial Dose</th>
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<td>5-Aminosalicylic acid</td>
<td>Oral</td>
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Table 02.

Treatment Approaches
Treatment in ulcerative colitis is individualized to the specific needs of the patient and alterations in treatment strategies are made according to the response attained. Nevertheless, we present a guide to the most common approaches used with our patients.

Mild Acute Relapsing Ulcerative Colitis
Mild disease is associated with four or fewer loose bowel movements daily with occasional blood, abdominal cramps, and, infrequently, tenesmus. Systemic symptoms are not present. For proctitis or proctosigmoiditis, symptomatic treatment with antidiarrheals, rectal steroids (or rectal 5-aminosalicylic acid [5-ASA]), and occasionally oral 5-ASA is recommended. Left-sided colitis or pancolitis is treated with rectal steroids and oral 5-ASA.

Moderate Acute Relapsing Ulcerative Colitis
In patients with moderate disease, bowel movements range from 4–8 daily with urgency, a nocturnal pattern, blood in the stool, abdominal discomfort, and some systemic symptoms such as weight loss, mild anemia and low-grade fever (less than 100°F). Proctitis or proctosigmoiditis is treated symptomatically (antidiarrheals, bulk agents). Rectal steroids (rectal 5-ASA) and oral 5-ASA are used in increasing doses. In left-sided or pancolitis, oral steroids are added and 5-ASA is used for maintenance therapy.
Severe Acute Relapsing Ulcerative Colitis

Severe attacks are characterized by the passage of six or more bloody stools daily accompanied by systemic symptoms such as fevers of 100°F or greater, weight loss, tachycardia, anemia with hemoglobin count of 10 g/dl or less, and hypoalbuminemia. For proctitis or proctosigmoiditis, double-dose rectal steroids (plus rectal 5-ASA) along with increased oral 5-ASA or oral or intravenous steroids, are recommended. In left-sided or pancolitis, no antidiarrheal medications are recommended. A combination of oral 5-ASA, rectal steroids, intravenous steroids, and intravenous antibiotics (i.e., ciprofloxacin and/or metronidazole) is recommended. In protracted cases, the addition of intravenous cyclosporine is considered. The usual dose is 4 mg/kg given in a four-hour intravenous infusion (2–6 pm) for a period of 5–7 days. Trough levels are followed (normal range 100–250 mg/dl) as well as renal (kidney) function while on intravenous cyclosporine. If there is no major improvement of symptoms within one week after the initiation of intravenous cyclosporine, the patient is usually referred for surgery.

Surgical Therapy

Surgery in ulcerative colitis should be reserved for those patients with refractory disease, complications associated with the medical therapy, or complications of colitis. Colectomy may be used in pediatric patients for amelioration of growth retardation in prepubescent children affected by ulcerative colitis. Current surgical alternatives include total proctocolectomy (Figure 16A) with Brooke ileostomy (Figure 16B), the intra-abdominal Koch pouch (Figure 16C), and restorative proctocolectomy with ileal pouch-anal anastomosis (Figure 16D).

![Figure 16. Surgical options for the treatment of ulcerative colitis; A, proctocolectomy; B, Brooke ileostomy; C, Koch pouch ileostomy; D, restorative proctocolectomy.](image1)

Elective colectomy cures ulcerative colitis and has a very low mortality rate (less than 1%). The procedure should almost always be a total colectomy (Figure 17A) with ileostomy or one of two internal ileal pouch alternatives. The Brooke ileostomy (standard) is a half-dollar–sized segment of terminal ileum that protrudes and is spouted from the right lower quadrant of the abdomen (Figure 17B). The patient attaches a double-faced adhesive ring to the skin and then to an opaque sack (which can be emptied) that collects the 750-1000 ml of material that the ileum produces daily (Figure 17C). Ostomy societies can be very helpful in adjusting to the inconvenience and psychological issues of an ileostomy.

![Figure 17. A, Proctocolectomy; B, Brooke ileostomy; C, side view with ileostomy bag.](image2)

The Koch pouch (continent) ileostomy is an alternative to the Brooke ileostomy. An internal reservoir is created from reshaped ileum with a nickel-sized nipple valve opening onto the lower abdominal wall. The patient catheterizes the pouch through a nipple valve to remove ileal contents. The main disadvantage of this approach is that the valve may become incontinent within 2–5 years in 25–30% of patients, necessitating surgical repair (Figure 18 A-C).
The most popular ileostomy alternative is the ileal pouch-anal anastomosis. The surgery involves creation of a new rectum from the small bowel and attaching the pouch of ileum to the anal canal (Figure 19). The pouch-anal anastomosis may be performed using a hand-sewn or stapled technique (Figure 20).

In patients with persistent disease activity or the development of dysplasia or cancer, a mucosectomy (stripping) may be performed before the anastomosis. Those who do not advocate anal stripping believe that preservation of a few centimeters of rectal mucosa produces better functional results (Figure 21).
In the patient with fulminant colitis, the colon may be removed first, leaving the creation of the pouch, restoration, and the removal of the rectum for a time when the patient has recovered from the colitis and is in better nutritional condition. This is a three-stage procedure, as a temporary ileostomy is made above the pelvic pouch to allow healing.

In patients with more chronic and stable disease, the procedure may be performed in two stages (with a temporary ileostomy). Select patients are candidates for a restorative proctocolectomy performed in a single step. After a temporary protective ileostomy is closed, patients can defecate through their anus. After one year, most patients have five bowel movements per day. Incontinence is uncommon, although some patients experience nocturnal soiling. Although pouchitis is a complication in 25% of patients, the ileoanal pouch is an acceptable and successful alternative to standard ileostomy.

Overview
The complications of ulcerative colitis can be divided into those that affect the colon and those that are extracolonic.

Toxic Megacolon
Overview
The most feared complication of ulcerative colitis is the development of toxic megacolon. It occurs as a result of extension of the inflammation beyond the submucosa into the muscularis, causing loss of contractility and ultimately resulting in a dilated colon. Dilatation of the colon is associated with a worsening of the clinical condition and development of fever and prostration.

Diagnosis
This diagnosis is based on radiographic evidence of colonic distention in addition to at least three of the four following conditions: fever higher than 38.6°C, neutrophil leukocytosis greater than 10,500 cells/mm³, heart rate greater than 120 beats/minute, and/or anemia. At least one sign of toxicity must also be present (dehydration, electrolyte disturbance, hypotension, or mental changes). Physical exam reveals a tender abdomen over the distribution of the colon. There may be rebound tenderness, abdominal distention, and hypoactive or absent bowel sounds.

Perforation
Colonic perforations are usually a complication of toxic megacolon. However, perforation can also present in severe ulcerative colitis even in the absence of toxic megacolon. Most perforations occur in the left colon, commonly in the sigmoid colon. Perforations tend to occur more often during the first episodes of colitis. Steroid therapy has been suggested to be a risk factor for colonic perforation, but this is controversial. Surgical management is indicated for perforation.

Radiography
X-rays of the abdomen reveal colonic dilation, usually maximal in the transverse colon, which tends to exceed 6 cm in diameter. Segments of the right and left colon
may also be dilated. Serial plain abdominal x-rays of the abdomen taken at 12–24-hour intervals are useful in following the clinical course.

Medical Therapy
The goal of medical therapy is to reduce the likelihood of perforation and to return the colon to normal motor activity. The patient should have nothing by mouth. A nasogastric tube is placed in the stomach for suction and decompression of the upper gastrointestinal tract. The use of the rolling technique, during which the patient lies on the abdomen for 10–15 minutes every 2 hours while awake, allows for passage of gas and easier decompression of the dilated colon. Intravenous fluids are given to replete water and electrolytes. Broad-spectrum antibiotic coverage is instituted in anticipation of peritonitis resulting from perforation. Intravenous steroids are usually administered in doses equivalent to more than 40 mg of prednisone per day. Close monitoring of the patient’s clinical condition is essential, and signs of deterioration, such as increasing abdominal girth, development of rebound tenderness, or hypotension, should prompt immediate action.

Surgical Therapy
Colectomy occurs in about 25% of patients and is required in almost 50% of patients with pancolitis. Surgical intervention is undertaken if the patient does not begin to show signs of improvement during the first 24–48 hours of medical therapy, as the risk of perforation increases markedly. Colectomy with creation of an ileostomy is the standard procedure, although single-stage proctocolectomy is done occasionally. If surgical therapy is performed before there is colonic perforation, the mortality is approximately 2%. In cases in which there has been bowel perforation, however, the mortality risk increases to 44%.

Strictures
Clinically relevant strictures are uncommon in ulcerative colitis. However, some degree of narrowing may be seen in approximately 12% of surgical specimens. Histologically, strictures present with hypertrophy and thickening of the muscularis mucosa without evidence of fibrosis. Strictures tend to occur late in the course of disease, usually 10–20 years after onset of disease. Most strictures occur in the sigmoid and rectum, with an approximate length of 2–3 cm. The most common presenting symptoms are diarrhea and fecal incontinence. Strictures have been associated with malignancy, and biopsy of the strictures is warranted. In fact, in patients with long-standing history of ulcerative colitis, a stricture should be considered potentially malignant.

Primary Sclerosing Cholangitis
Primary sclerosing cholangitis is a chronic cholestatic liver disease characterized by fibrosing inflammation of extra- and intrahepatic bile ducts. It is frequently associated with ulcerative colitis (Figure 24). Patients may have symptoms of fatigue, pruritis, abdominal pain, fever, or jaundice. This usually appears in men after 10–15 years of very mild, even subclinical, pancolitis, and may necessitate liver transplantation in some patients.

![Figure 23. A, Primary sclerosing cholangitis with typical stricture and dilation pattern; B, Cholangiogram (ERCP image)](image)