History
A 23-year-old man with inflammatory bowel disease diagnosed in May experienced a deteriorating clinical course despite maximal medical therapy. He was admitted to the hospital in August with abdominal pain, weight loss of 60 lb (27 kg), and 8–10 episodes per day of diarrhea with intermixed blood and mucus. His family history revealed two maternal cousins with inflammatory bowel disease. At hospital admission, he was mildly tachycardic (104 beats per minute) but normotensive and afebrile. A physical examination revealed abdominal distention without rebound or guarding. Pertinent laboratory values included an elevated white blood cell count of 11,700 cells per microliter and a left shift, as well as a hemoglobin level of 10.9 g/dL, indicative of anemia. The patient was placed on a regimen of bowel rest with intravenous hydration, total parenteral nutrition, hydrocortisone, antibiotics, and cyclosporine. Computed tomography (CT) of the abdomen and pelvis was performed. By day 9 of hospitalization, the frequency of episodes of diarrhea had increased to 12 times per day. The patient was febrile to 38°C, tachycardic to 125 beats per minute, and anemic, with a hemoglobin level of 8.5 g/dL. Numerous electrolyte abnormalities were present, including hypokalemia, hypophosphatemia, and hypocalcemia. Supine and upright radiographs of the abdomen demonstrated stable dilatation of the transverse colon without free intraperitoneal air. On day 10 of the patient’s hospital stay, a subtotal colectomy with ileostomy was performed.

Imaging Findings
CT of the abdomen and pelvis was performed after the administration of oral and intravenous contrast material. The topogram revealed nodular areas of high attenuation overlying an apparently ahausstral transverse colon, which was dilated to 8 cm (Fig 1). In the right colon, CT images revealed dilatation, circumferential mural thickening, an irregular mucosal surface (Fig 2), and a target sign (Fig 3). Scattered gas bubbles adjacent to the dependent wall of the cecum suggested pneumatisis, but no portal venous gas was
present (Fig 3). Pseudopolyps on the wall of the transverse colon extended into the lumen (Fig 4). The descending and sigmoid portions of the colon were partly collapsed but appeared to have thickened walls. Dilated blood vessels near the sigmoid colon and high attenuation within adjacent fat indicated inflammation and hyperemia (Fig 5). The small bowel and its mesentery were spared. A moderate amount of fluid was present throughout the abdomen and pelvis, but there was no free air.

**Pathologic Evaluation**

The subtotal colectomy specimen was 78 cm long. The right colon and transverse colon portions demonstrated marked dilatation. Mucosal ulceration and polypoid lesions, some of which were covered by tan and yellow exudates, were obvious at gross inspection (Fig 6). At microscopy, extensive ulceration was visible that extended to the level of the inflamed muscularis propria. Protruding from this base were pseudopolyps consisting of residual mucosa and submucosa, many of which were covered by granulation tissue (Fig 7a). There was neither gross nor microscopic evidence of air within the colonic mucosa or submucosa. Dilated blood vessels near the sigmoid colon and high attenuation within adjacent fat indicated inflammation and hyperemia (Fig 5). The small bowel and its mesentery were spared. A moderate amount of fluid was present throughout the abdomen and pelvis, but there was no free air.
The diagnosis was ulcerative colitis. Focal eruptions of pseudomembrane indicated a superimposed *Clostridium difficile* infection (Fig 7b).

**Discussion**

Inflammatory bowel disease refers to a group of disorders characterized by intestinal inflammation, extra-intestinal manifestations, and a relapsing course (1). Although ulcerative colitis and Crohn disease account for the majority of cases of inflammatory bowel disease, indeterminate colitis, an entity that demonstrates overlapping clinical, imaging, and histologic features, represents up to 6% of cases (2).

The etiology of ulcerative colitis is unknown, though studies demonstrating a more frequent occurrence within members of the same family (3) as well as within northern and urban areas (4) suggest an interaction between genetic and environmental factors. The annual North American incidence is 2–14 per 100,000 persons in the general population (5), with males affected more often than females (1.3:1) (5–7). Most individuals with the condition are between the ages of 15 and 40 years at the time of diagnosis (6).

The presence of ulcerative colitis or Crohn disease may be suspected in patients with persistent diarrhea, urgency of defecation, and tenesmus, often associated with fever, pain, and weight loss (8). Because these symptoms are not specific to inflammatory bowel disease, the initial work-up should include stool studies and biopsy to exclude infectious causes (9). When imaging is required, colonoscopy is preferred, as it permits both direct

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**Figure 6.** (a) Photograph shows continuous regions of ulceration and pseudopolyps throughout the subtotal colectomy specimen, with the most severe changes evident in the ascending and transverse colon portions, and dilatation indicative of megacolon. (b) Close-up photograph of the ascending colon shows pseudopolyps with a partial overlay of fibrinous exudate, as well as ulceration of the adjacent mucosa.

**Figure 7.** (a) Photomicrograph (whole mount; original magnification, ×2; hematoxylin-eosin stain) of the resected specimen shows a pseudopolyp (arrow) that extends from a base of denuded muscularis propria (arrowhead). Inflammatory cells within the muscularis propria indicate extensive inflammation. The surface of the pseudopolyp contains regenerative glands. (b) Photomicrograph (original magnification, ×10; hematoxylin-eosin stain) shows a volcano-like eruption of pseudomembrane (arrow) that contains epithelial debris, fibrin, mucus, and neutrophils.
visualization of the colonic mucosa and tissue sampling (8,10). Contrast-enhanced luminal radiography (a barium enema study) is essential, however, when anatomic barriers such as colonic strictures prevent adequate endoscopic examination or when disease is suspected within the small intestine (10). The double-contrast barium enema and small-bowel barium follow-through examination can demonstrate fine mucosal detail and provide valuable information on luminal dis- tensibility, stricturing, and fistula formation (10). In fact, the small-bowel series remains the test of choice for evaluation of the small intestine (1). In cases of severe colitis, both colonoscopy and the barium enema study are contraindicated because of an increased risk of perforation (10). In addition, neither modality is capable of demonstrating the transmural extent of disease. Therefore, CT has emerged as a valuable tool of evaluation for intraperitoneal complications, including abscess, fistula, and perforation (1,10,11).

Distinguishing ulcerative colitis from Crohn disease is an important challenge, as the complications, management, and prognosis of the diseases differ (2). Crohn disease can affect the length of the digestive tract in a discontinuous fashion, usually most severely at the ileum and cecum and with relative sparing of the rectum (10,12,13). Perianal disease is common (14). The bowel wall can be affected asymmetrically, with retraction at the mesenteric margin and sacculation of the antimesenteric edge (14). Because the inflammatory process is typically transmural, ulceration with a nodular or cobblestone appearance and with fissures, fistulas, and abscesses is common (13,14).

In contrast, ulcerative colitis is generally a superficial inflammatory process that affects the colonic mucosa (13). Rectal involvement is present in 95% of cases, with variable degrees of contiguous, circumferential, and proximal extension throughout the large intestine (8). Small-bowel disease is uncommon. Therefore, a small-bowel barium series may assist in differentiating ulcerative colitis from Crohn disease (8). A minority of patients with pancolonic ulcerative colitis, however, demonstrate edematous changes of the terminal ileum. Also known as backwash ileitis, this process is distinguished from Crohn disease by the presence of a patulous ileocecal valve and the absence of ulceration (9,10,14). Fistulas and abscesses may be present but are uncommon (11,15).

Mural thickening is a common manifestation of inflammatory bowel disease; in general, however, ulcerative colitis produces less wall thickening than does Crohn disease (10,15). The earliest expressions of inflammation are hyperemia and altered mucin production. These alterations are responsible for the grainy mucosal appearance noted on radiographs from barium studies in patients with acute ulcerative colitis (14,15). As inflammation worsens, the colonic wall thickens, and thumbnail-like indentations form that are visible on plain radiographs and images from barium studies (12). When edema occurs disproportionately in the various layers of the bowel wall, as in the case described in this article, a series of concentric rings of varied attenuation known as the target sign can be seen on intravenous contrast material–enhanced CT images (11,14).

Eventual erosion through the mucosa and submucosa results in a characteristic button-shaped ulcer that can be seen on images from barium studies (12). As adjacent ulcers coalesce, extensive regions of the mucosa are undermined and slough off, leaving islands of residual tissue that extend into the colonic lumen (12). The mushroom-shaped mucosal remnants, or pseudopolyps, may be visible when they are outlined by air on plain radiographs and CT images (11,12).

Like this patient, an estimated 15%–20% of individuals with ulcerative colitis develop a fulminating form (14) that is characterized by severe symptoms and by inflammation that extends deep beneath the colonic mucosa (12,16). Damage to the muscularis propria results in colonic dilatation and loss of haustra (16). These symptoms are most prominent in nondependent loops of bowel, such as the transverse colon, into which air can rise in a supine patient (14,16). Serosal irritation and ascites are rare and may be related either to ulcerative colitis or to superimposed processes such as C difficile infection (11). Toxic megacolon, a potentially fatal complication that developed in this individual, is seen in less than 5% of patients (14,16) and is characterized by both nonobstructive dilatation of the colon to at least 6 cm and evidence of systemic toxicity (17). Although toxic megacolon may occur at any point during the course of ulcerative colitis (14,16), it has been reported that 30% of cases develop within 3 months of diagnosis (16). Morbidity may result
from a number of factors, including electrolyte disturbance, fluid loss, hemorrhage, and perforation (16).

In patients with a less severe course of disease, a so-called lead-pipe colon may be depicted on images from barium studies. This condition may occur for a number of reasons in patients with chronic ulcerative colitis. First, mucosal regeneration may lead not only to filiform polyp formation but also to hypertrophy of the muscularis mucosae (11,12). Contraction of the enlarged muscle layer gives the colon a narrowed, ahastral, and foreshortened appearance (12,14). Second, structures may compromise luminal distensibility (12). Third, narrowing may be caused by fat deposition within the submucosal layer of the bowel wall, particularly in the rectum (11,14). Last, extramural fat proliferation, particularly in a perirectal location, can cause luminal narrowing and a characteristic widening of the presacral space (15).

Chronic ulcerative colitis is associated with an increased colorectal cancer risk that depends on the duration and extent of disease and that ranges from 0.5% to 1.0% per year after 10 years of universal disease. Therefore, surveillance is recommended (8). A high degree of suspicion is necessary, as carcinoma associated with ulcerative colitis can form plaquelike, infiltrating, or scirrhous tumors that may simulate benign strictures (12,14).

The most suitable therapy for each individual is determined by the severity of the symptoms and the extent of disease. Medical therapy reduces inflammation but does not cure the underlying process; therefore, many patients experience waxing and waning symptoms over time (13). In contrast to medical therapy, surgery cures the intestinal symptoms of ulcerative colitis but incurs new risks and requires alterations in lifestyle (13). Elective surgery is contemplated for those patients with long-standing ulcerative colitis who develop medically intractable symptoms, serious drug-related side effects, dysplasia, or malignancy (8,13). In the setting of exsanguinating hemorrhage, perforation, or fulminant colitis that is unresponsive to maximal medical therapy, an emergent colectomy is required (8,13).

Because of severe symptoms and progression to toxic megacolon, the patient in this case underwent the most common emergency procedure, total abdominal colectomy with Hartmann pouch and an end ileostomy. This surgery is associated with a lower risk of bleeding and pelvic nerve injury than emergent proctocolectomy, and it leaves the rectum in place for use in a future continence-restoration procedure (13). He underwent a completion proctocolectomy and ileal pouch anal anastomosis without complication 3 months later.

References