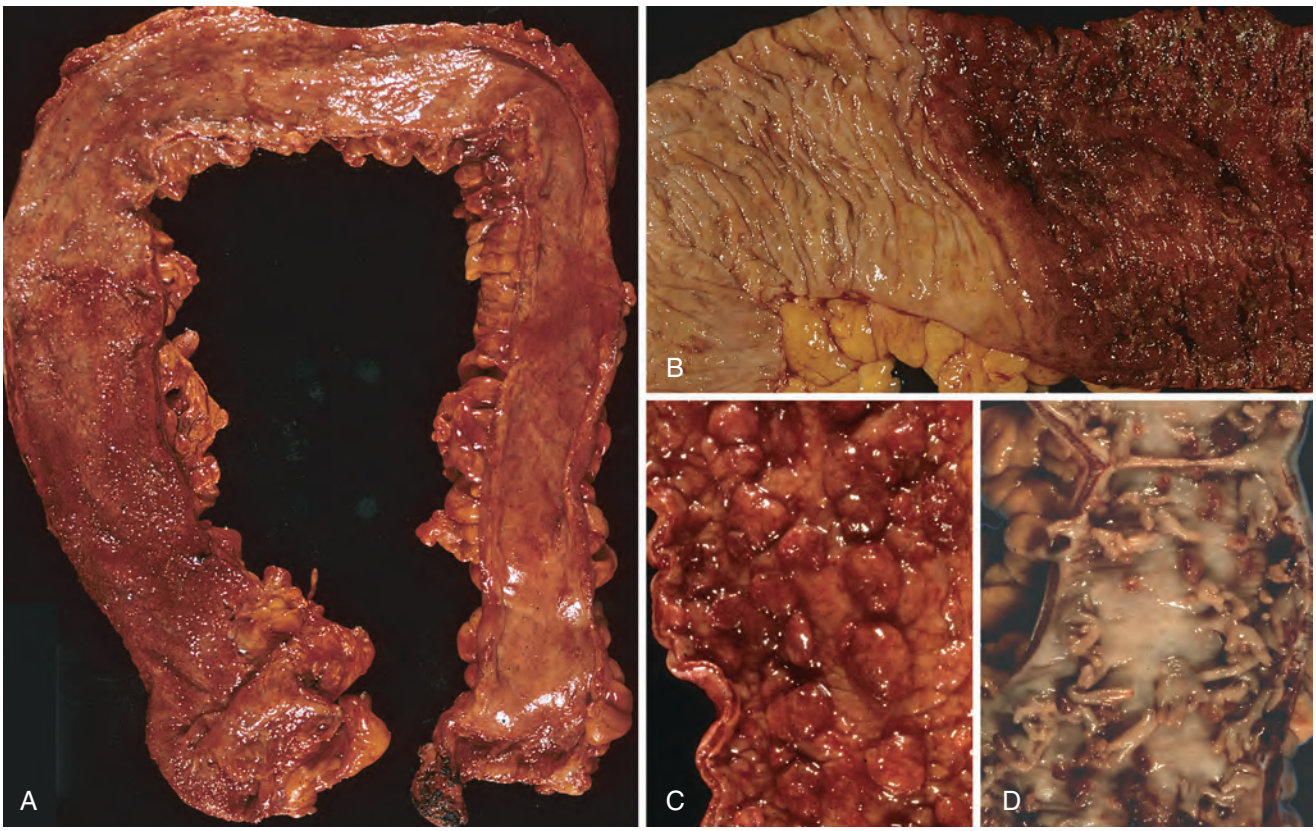

Figure 17-36 Gross pathology of ulcerative colitis. **A,** Total colectomy with pancolitis showing active disease, with red, granular mucosa in the cecum (left) and smooth, atrophic mucosa distally (right). **B,** Sharp demarcation between active ulcerative colitis (right) and normal mucosa (left). **C,** Inflammatory polyps. **D,** Mucosal bridges.

definitive diagnosis is not possible in approximately 10% of IBD patients. These cases, termed indeterminate colitis, do not involve the small bowel and have colonic disease in a continuous pattern that would typically indicate ulcerative colitis. However, patchy histologic disease, fissures, a family history of Crohn disease, perianal lesions, onset after initiating use of cigarettes, or other features that are not typical of ulcerative colitis may prompt more detailed endoscopic, radiographic, and histologic examination. Serologic studies can be useful in these cases because perinuclear anti-neutrophil cytoplasmic antibodies are found in 75% of individuals with ulcerative colitis but only 11% with Crohn disease. In contrast, ulcerative colitis patients tend to lack antibodies to *Saccharomyces cerevisiae*, which are often present in those with Crohn disease. However, even the serologic results can be ambiguous in cases that are indeterminate on clinical grounds. Despite diagnostic uncertainty, extensive overlap in medical management of ulcerative colitis and Crohn disease allows patients carrying a diagnosis of indeterminate colitis to be treated effectively.

Colitis-Associated Neoplasia

One of the most feared long-term complications of ulcerative colitis and colonic Crohn disease is the development of neoplasia. The risk of dysplasia is related to several factors:

- *Duration of the disease.* Risk increases sharply 8 to 10 years after disease onset.
- *Extent of the disease.* Patients with pancolitis are at greater risk than those with only left-sided disease.
- *Nature of the inflammatory response.* Greater frequency and severity of active inflammation (characterized by the presence of neutrophils) confers increased risk.

To facilitate early detection of neoplasia, patients are typically enrolled in surveillance programs approximately 8 years after diagnosis of IBD. The major exception to this is patients with IBD and primary sclerosing cholangitis, who have an even greater risk of developing cancer and are generally enrolled for surveillance at the time of diagnosis. Surveillance requires regular and extensive mucosal biopsies, making it a costly practice. Research efforts are therefore focused on discovery of molecular markers of dysplasia.

The goal of surveillance biopsies is to identify dysplastic epithelium, which is a precursor to colitis-associated carcinoma. Dysplasia can develop in flat areas of mucosa that are not grossly recognized as abnormal. Thus, advanced endoscopic imaging techniques including chromoendoscopy and confocal endoscopy are beginning to be used to increase the sensitivity of detection. IBD-associated dysplasia is classified histologically as low grade or high grade (Fig. 17-38*A, B*) and may be multifocal. High-grade dysplasia may be associated with invasive carcinoma at the same site (Fig. 17-38*C*) or elsewhere in the colon and, therefore, often prompts colectomy. Low-grade dysplasia may be treated with colectomy or followed closely, depending on a variety of factors including patient age and the number of dysplastic foci present. Colonic