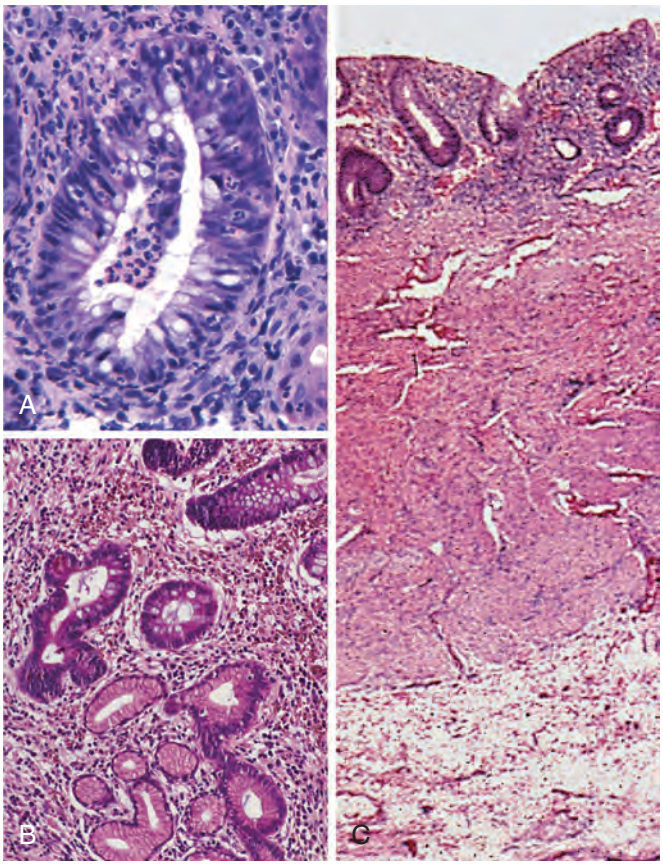

Figure 17-37 Microscopic pathology of ulcerative colitis. **A,** Crypt abscess. **B,** Pseudopyloric metaplasia (bottom). **C,** Disease is limited to the mucosa. Compare to Figure 17-35*C*.

adenomas (discussed later) also occur in IBD patients, and in some cases these may be difficult to differentiate from a polypoid focus of IBD-associated dysplasia.

Other Causes of Chronic Colitis

Diversion Colitis

Surgical treatment of ulcerative colitis, Hirschsprung disease and other intestinal disorders sometimes require creation of a temporary or permanent ostomy and a blind distal segment of colon, from which the normal fecal flow is diverted. Colitis can develop within the diverted segment, particularly in ulcerative colitis patients. Besides mucosal erythema and friability, the most striking feature of diversion colitis is the development of numerous mucosal lymphoid follicles (Fig. 17-39*A*). Increased numbers of lamina propria lymphocytes, monocytes, macrophages, and plasma cells may also be present. In severe cases the histopathology may resemble IBD and include crypt abscesses, mucosal architectural distortion, or, rarely, granulomas. The mechanisms responsible for diversion colitis are not well understood, but changes in the luminal microbiota and diversion of the fecal stream that provides nutrients to colonic epithelial cells have been proposed. Consistent with this, enemas containing short-chain fatty acids, a product of bacterial digestion in the colon and an important energy source for colonic epithelial cells, can promote mucosal recovery in some cases. The ultimate cure is reanastomosis of the diverted segment.

Microscopic Colitis

Microscopic colitis encompasses two entities, *collagenous colitis* and *lymphocytic colitis*. These idiopathic diseases both present with chronic, nonbloody, watery diarrhea without weight loss. Radiologic and endoscopic studies are typically normal. Collagenous colitis, which occurs primarily in middle-aged and older women, is characterized by the presence of a dense subepithelial collagen layer, increased numbers of intraepithelial lymphocytes, and a mixed inflammatory infiltrate within the lamina propria (Fig. 17-39*B*). Lymphocytic colitis is histologically similar, but the subepithelial collagen layer is of normal thickness and the increase in intraepithelial lymphocytes is greater, frequently exceeding one T lymphocyte per five colonocytes (Fig. 17-39*C*). Lymphocytic colitis shows a strong association with celiac disease and autoimmune diseases, including Graves disease, rheumatoid arthritis, and autoimmune or lymphocytic gastritis.

Graft-Versus-Host Disease

Graft-versus-host disease occurs following hematopoietic stem cell transplantation. The small bowel and colon are involved in most cases. Although graft-versus-host disease is secondary to donor T cells targeting antigens on the recipient's GI epithelial cells, the lamina propria lymphocytic infiltrate is typically sparse. Epithelial apoptosis,

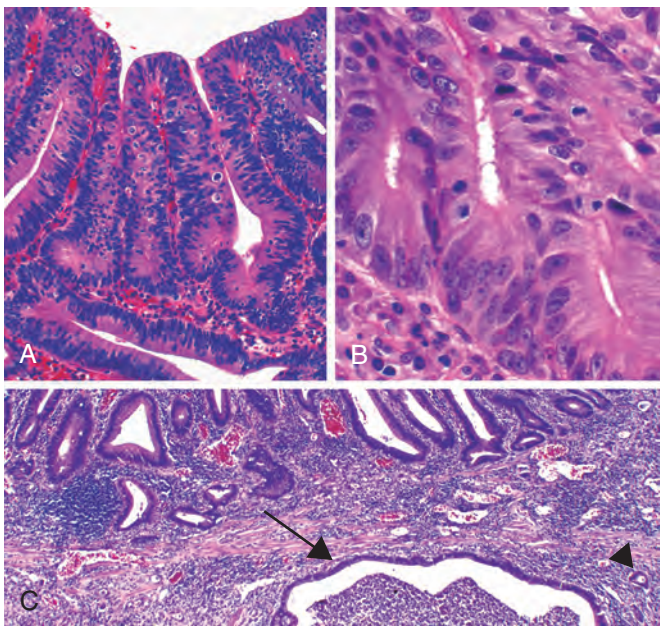

Figure 17-38 Colitis-associated dysplasia. **A,** Dysplasia with extensive nuclear stratification and marked nuclear hyperchromasia. **B,** Cribriform glandular arrangement in high-grade dysplasia. **C,** Colectomy specimen with high-grade dysplasia on the surface and underlying invasive adenocarcinoma. A large cystic, neutrophil-filled space lined by invasive adenocarcinoma is apparent *(arrow)* beneath the muscularis mucosae. Also seen are small invasive glands *(arrowhead)*.