Clinical Features. Most individuals with diverticular disease remain asymptomatic throughout their lives. However, about 20% of individuals with diverticuli develop manifestations of diverticular disease, such as intermittent cramping, continuous lower abdominal discomfort, constipation, distention, or a sensation of never being able to completely empty the rectum. Patients sometimes experience alternating constipation and diarrhea that can mimic IBS. Occasionally there may be minimal chronic or intermittent blood loss, and, rarely, massive hemorrhage. When present, bleeding is macroscopically visible in the stools. Whether a high-fiber diet prevents such progression or protects against diverticulitis is unclear, but diets supplemented with fiber may provide symptomatic improvement. Even when diverticulitis occurs, it most often resolves spontaneously and relatively few patients require surgical intervention.

KEY CONCEPTS

- **Irritable bowel syndrome (IBS)** is characterized by chronic, relapsing abdominal pain, bloating, and changes in bowel habits without obvious gross or histologic pathology. The pathogenesis of IBS is not defined, but includes contributions by psychologic stressors, diet, the gut microbiome, abnormal GI motility, and increased enteric sensory responses to gastrointestinal stimuli.
- **Inflammatory bowel disease (IBD)** is an umbrella term for **ulcerative colitis** and **Crohn disease**. **Indeterminate colitis** is used for cases of IBD without definitive features of either ulcerative colitis or Crohn disease.
- **Ulcerative colitis is limited to the colon**, is **continuous from the rectum**, and ranges from only rectal disease to pancolitis; neither skip lesions nor granulomas are present.
- **Crohn disease** most commonly affects the **terminal ileum and cecum**, but any site within the gastrointestinal tract can be involved; **skip lesions** are common and **noncaseating granulomas** also occur.
- Both forms of IBD typically present in the **teens and early 20s** and are associated with **extraintestinal manifestations**.
- IBD is thought to arise from a combination of alterations in host interactions with intestinal microbiota, intestinal epithelial dysfunction, and aberrant mucosal immune responses. Molecular analyses have identified more than 160 IBD-associated genes, of which the function of only a few is understood.
- The risk of colonic **epithelial dysplasia and adenocarcinoma** is increased in IBD patients who have had colonic disease for more than 8 to 10 years.
- The two forms of microscopic colitis, **collagenous colitis** and **lymphocytic colitis**, both cause chronic watery diarrhea. The intestines are grossly normal, and the diseases are identified by their characteristic histologic features.
- **Diverticular disease** of the sigmoid colon is common in western populations older than age 60. The causes include low fiber diets, colonic spasm, and the unique anatomy of the colon. Inflammation of diverticula, **diverticulitis**, affects a minority of those with **diverticulosis**, but can cause perforation in its most severe form.

Polyps

Polyps are most common in the colo-rectal region but may occur in the esophagus, stomach, or small intestine. Most, if not all, polyps begin as small elevations of the mucosa. These are referred to as sessile, a term borrowed from botanists who use it to describe flowers and leaves that grow directly from the stem without a stalk. As sessile polyps enlarge, proliferation of cells adjacent to the mass and the effects of traction on the luminal protrusion, may combine to create a stalk. Polyps with stalks are termed pedunculated. In general, intestinal polyps can be classified as nonneoplastic or neoplastic in nature. The most common neoplastic polyp is the adenoma, which has the potential to progress to cancer. The nonneoplastic polyps can be further classified as inflammatory, hamartomatous, or hyperplastic.

Hyperplastic Polyps

Colonic hyperplastic polyps are benign epithelial proliferations that are typically discovered in the sixth and seventh decades of life. The pathogenesis of hyperplastic polyps is incompletely understood, but they are thought to result from decreased epithelial cell turnover and delayed shedding of surface epithelial cells, leading to a "piling up" of goblet cells and absorptive cells. It is now appreciated that these lesions are without malignant potential. **Their chief significance is that they must be distinguished from sessile serrated adenomas, that are histologically similar but have malignant potential, as described later**. It is also important to remember that epithelial hyperplasia can occur as a nonspecific reaction adjacent to or overlying any mass or inflammatory lesion and, therefore, can be a clue to the presence of an adjacent, clinically important lesion.

MORPHOLOGY

Hyperplastic polyps are most commonly found in the left colon and are typically less than 5 mm in diameter. They are smooth, nodular protrusions of the mucosa, often on the crests of mucosal folds. They may occur singly but are more frequently multiple, particularly in the sigmoid colon and rectum. Histologically, hyperplastic polyps are composed of mature goblet and absorptive cells. The delayed shedding of these cells leads to crowding that creates the serrated surface architecture that is the morphologic hallmark of these lesions (Fig. 17-41). Serration is typically restricted to the upper third, or less, of the crypt.

Inflammatory Polyps

Polyps that form as part of the solitary rectal ulcer syndrome are examples of purely inflammatory lesions. Patients present with a clinical triad of rectal bleeding, mucus discharge, and an inflammatory lesion of the anterior rectal wall. The underlying cause is impaired relaxation of the anorectal sphincter that creates a sharp angle at the anterior rectal shelf and leads to recurrent abrasion and ulceration of the overlying rectal mucosa. An inflammatory polyp may ultimately form as a result of chronic cycles of injury and healing. Entrapment of this polyp in