Diseases of the intestines

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Diseases of the intestines

Intestinal obstruction

Vascular disorders

Malabsorptive diseases and infections

Inflammatory bowel disease.

Polyps and neoplastic diseases

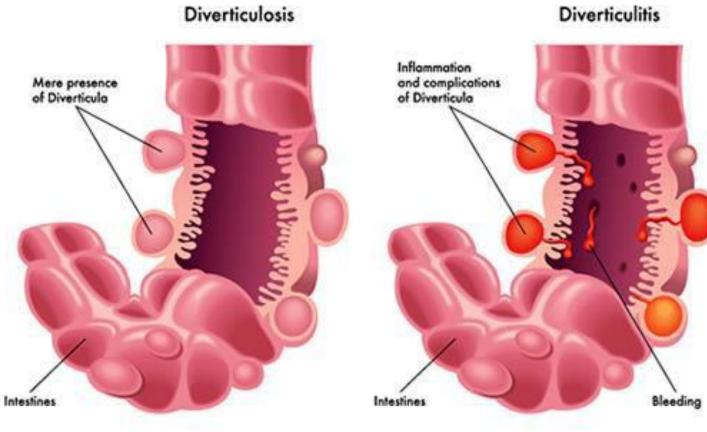
INFLAMMATORY INTESTINAL DISEASE

- Sigmoid Diverticulitis
- Chronic Inflammatory bowel diseases (CIBD)
- Crohn disease
- -Ulcerative colitis

Sigmoid Diverticulitis

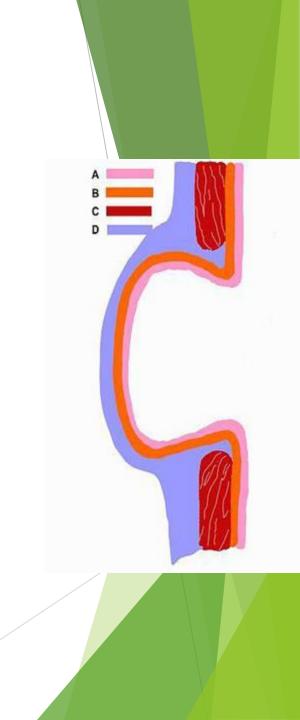
♪ In general, diverticular disease refers to acquired pseudodiverticular outpouchings of the colonic mucosa and submucosa.

- Diverticula generally are multiple, and the condition is referred to as diverticulosis.
- Colonic diverticula tend to develop under conditions of elevated intraluminal pressure in the sigmoid colon.
- J>High luminal pressures may be generated by exaggerated peristaltic contractions, with spasmodic sequestration of bowel segments that may be exacerbated by diets low in fiber and constipation, which reduce stool bulk.



MORPHOLOGY

- Flasklike outpouchings
- □ Mostly in sigmoid colon.
- □ Thin wall (atrophic mucosa, compressed submucosa)
- □ Attenuated or absent muscularis.
- Obstruction leads to diverticulitis.
- □ Risk of perforation.
- Recurrent diverticulitis leads
 to strictures.



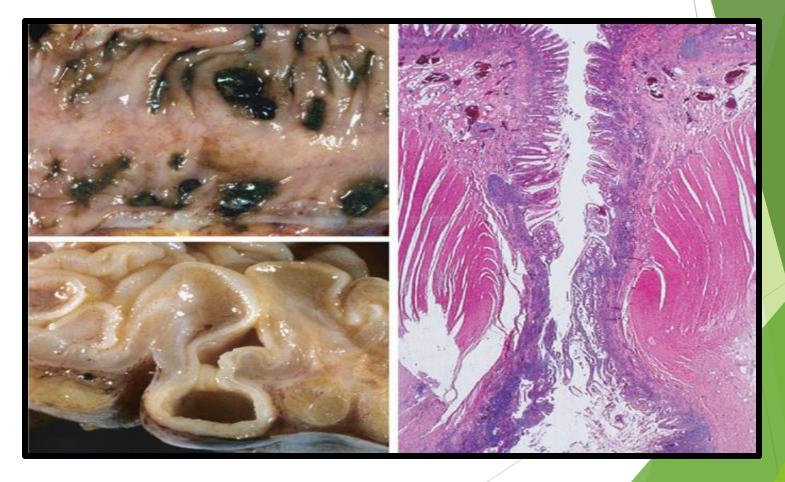


♪Sigmoid diverticular disease.

J>A, Stool-filled diverticula are regularly arranged.

1> B, Cross-section showing the outpouching of mucosa beneath the muscularis propria.

C, Low-power photomicrograph of a sigmoid diverticulum showing protrusion of the mucosa and submucosa through the muscularis propria.





COMPLICATIONS

J>Obstruction of diverticula leads to inflammatory changes, producing diverticulitis and peridiverticulitis.

Decause the wall of the diverticulum is supported only by the muscularis mucosa and a thin layer of subserosal adipose tissue, inflammation and increased pressure within an obstructed diverticulum can lead to perforation.

Clinical Features

• Mostly asymptomatic.

□ Intermittent lower abdominal pain

□ Constipation or diarrhea.

Тх

□ High fiber diet.

Antibiotics in diverticulitis.

Surgery.

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is a chronic condition resulting from inappropriate mucosal immune activation. IBD encompasses two major entities, Crohn disease and ulcerative colitis.

The distinction between ulcerative colitis and Crohn disease is based, in large part, on the distribution of affected sites and the morphologic expression of disease at those site.

Inflammatory Bowel Disease

- Ulcerative colitis is limited to the colon and rectum and extends only into the mucosa and submucosa.
- By contrast, Crohn disease, which also has been referred to as regional enteritis (because of frequent ileal involvement), may involve any area of the gastrointestinal tract and frequently is transmural

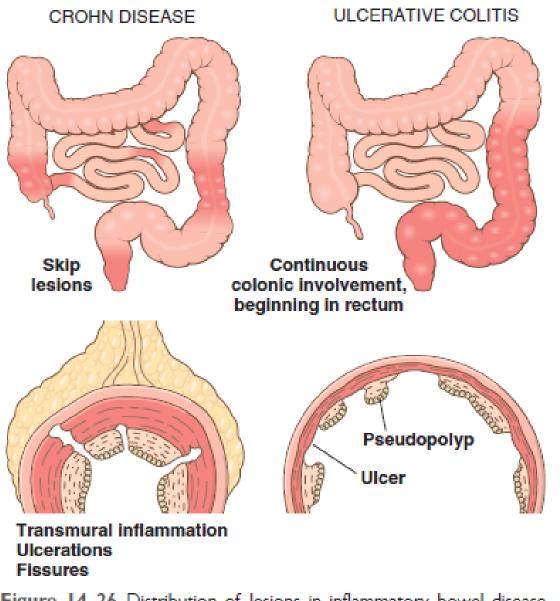


Figure 14–26 Distribution of lesions in inflammatory bowel disease. The distinction between Crohn disease and ulcerative colitis is based primarily on morphology.

Epidemiology

 Both Crohn disease and ulcerative colitis are more common in females and frequently present during adolescence or in young adults.
 Geographic variation.

- J>Hygiene hypothesis: childhood exposure to environmental microbes prevents excessive immune system reactions.
- ♪ Firm evidence is lacking!!!.

PATHOGENESIS

J>The cause(s) of IBD remains uncertain.

Combined effects.

Genetic factors.

□ Alterations in host interactions with intestinal microbiota

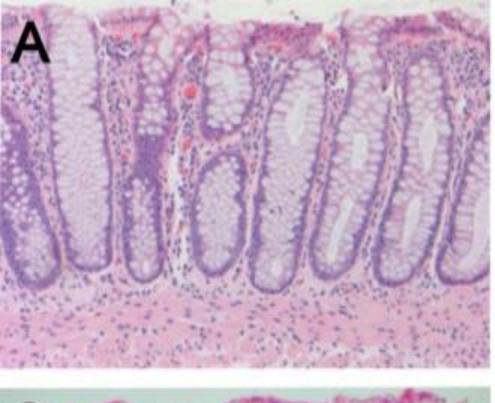
- □ Intestinal epithelial dysfunction.
- □ Aberrant mucosal immune responses
- □ Altered composition of the gut microbiome.

MORPHOLOGY OF Crohn Disease

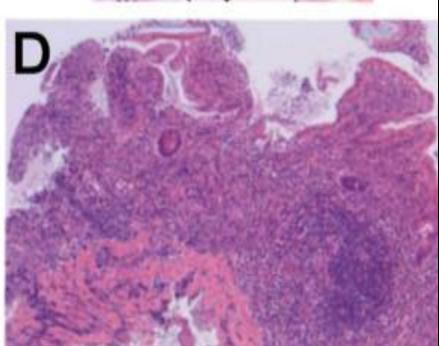
The most common sites involved by Crohn disease at presentation are the terminal ileum, ileocecal valve, and cecum.

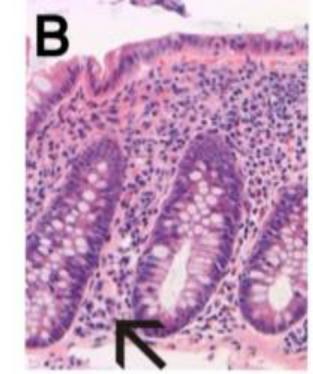
- The presence of multiple, separate, sharply delineated areas of disease, resulting in skip lesions, is characteristic of Crohn disease.
- ♪Strictures are common.
- Sparing of interspersed mucosa results in a coarsely textured, cobblestone appearance in which diseased tissue is depressed below the level of normal mucosa.
- J>Fissures frequently develop between mucosal folds and may extend deeply to become sites of perforation or fistula tracts.
- The intestinal wall is thickened..
- In cases with extensive transmural disease, mesenteric fat frequently extends around the serosal surface (creeping fat).

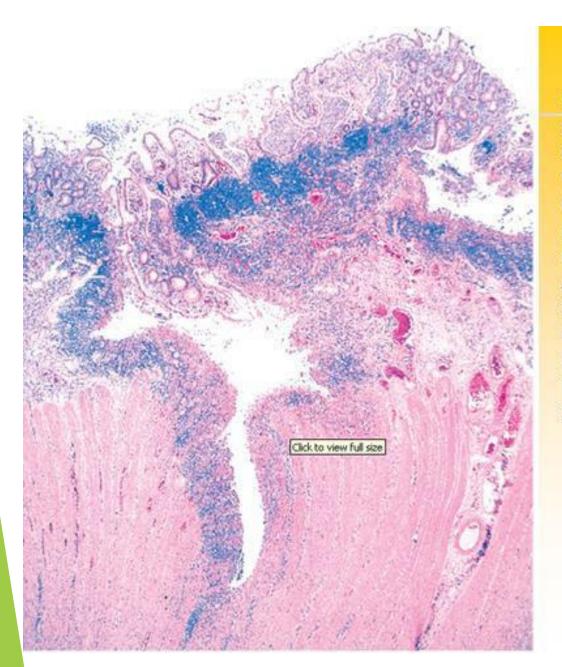




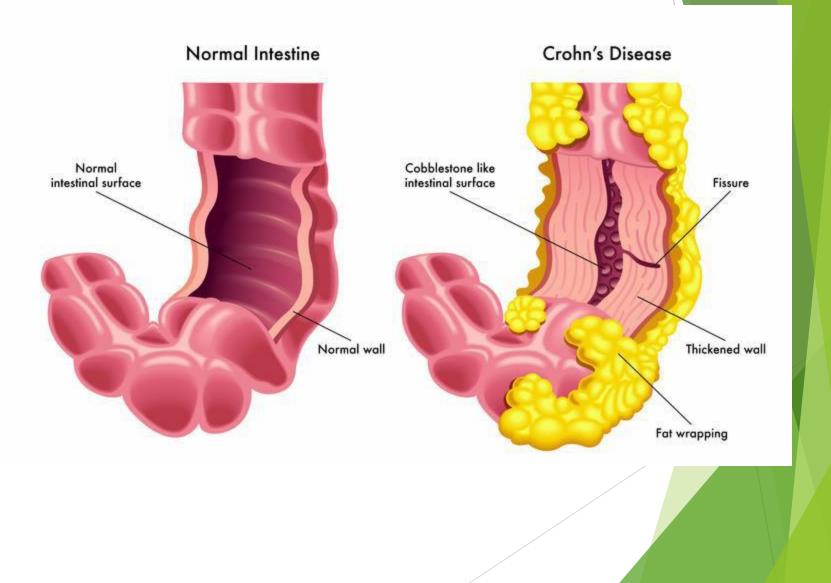


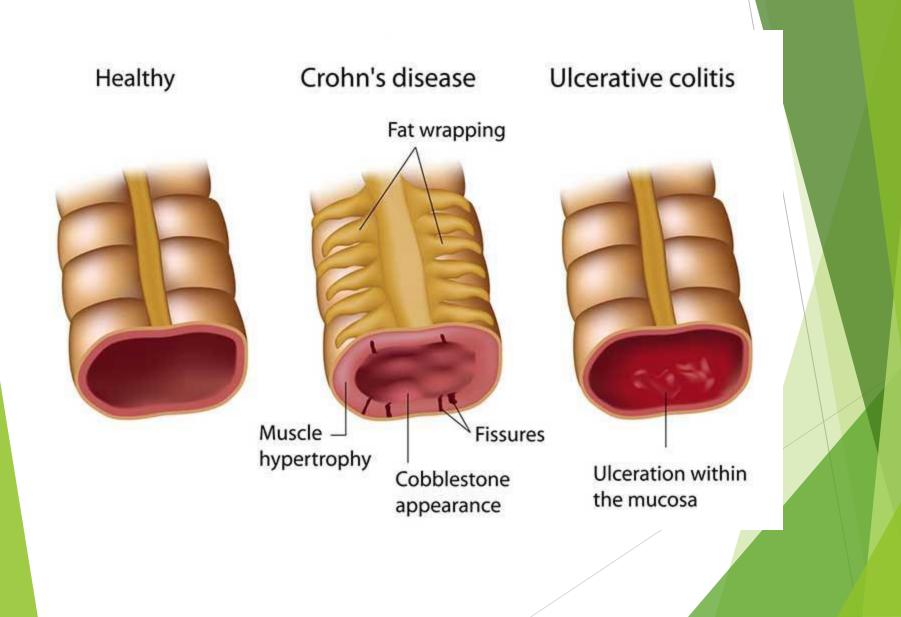






Crohn disease of the colon showing a deep fissure extending into the muscle wall, a second, shallow ulcer (upper right), and relative preservation of the intervening mucosa. Abundant lymphocyte aggregates are present, evident as dense blue patches of cells at the interface between mucosa and submucosa





Cobblestone appearance



The microscopic features of active Crohn disease include:

1- Abundant neutrophils that infiltrate -and damage crypt epithelium.

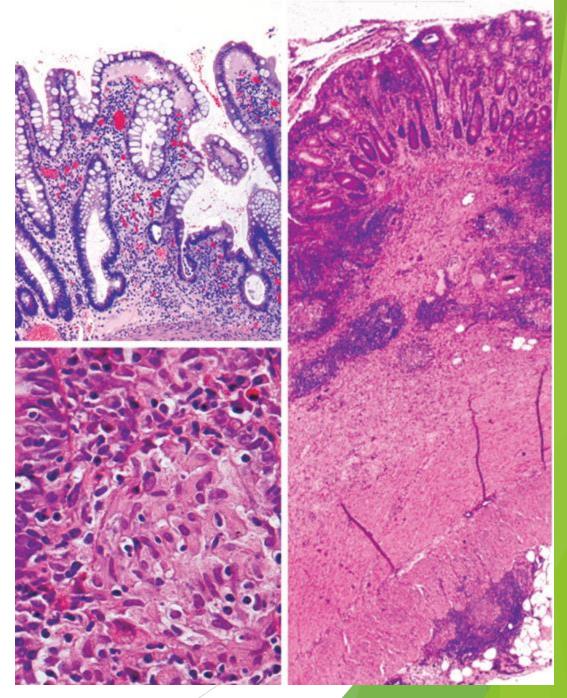
2- Clusters of neutrophils within a crypt are referred to as a **crypt abscess** and often are associated with crypt destruction.

3- Repeated cycles of crypt destruction and regeneration

lead to distortion of mucosal architecture; the normally straight and parallel crypts take on bizarre branching shapes and unusual orientations to one another.

4- **Noncaseating granulomas** , a hallmark of Crohn disease.

5- Paneth cell metaplasia



Clinical Features

The clinical manifestations of Crohn disease are extremely variable. *In most patients, disease begins with intermittent attacks of relatively mild diarrhea, fever, and abdominal pain.*

Iron deficiency anemia, nutrient malabsorption, or malabsorption of vitamin B12 and bile salts may developed.

I> Extraintestinal manifestations of Crohn disease include uveitis, migratory polyarthritis, sacroiliitis, ankylosing spondylitis, erythema nodosum, and clubbing of the fingertips, any of which may develop before intestinal disease is recognized.

The risk of colonic adenocarcinoma is increased in patients with long-standing colonic Crohn disease.

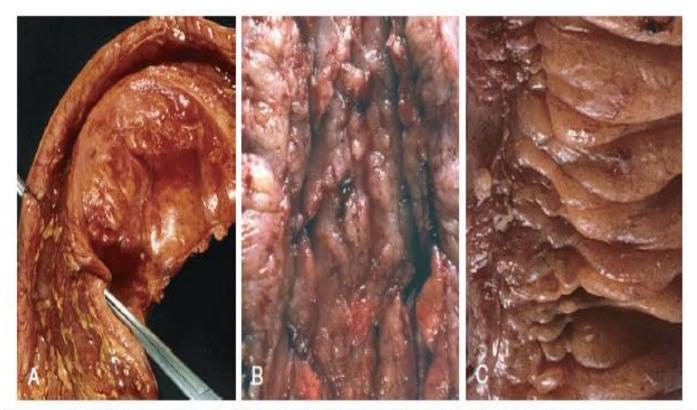


Figure 14-28 Gross pathology of Crohn disease. A, Small intestinal stricture. B, Linear mucosal ulcers and thickened intestinal wall. C, Creeping fat.



Ulcerative Colitis

J>Ulcerative colitis is closely related to Crohn disease.

∴>However, ulcerative colitis is limited to the colon and rectum.

Some extraintestinal manifestations of ulcerative colitis overlap with those of Crohn disease, including migratory polyarthritis, sacroiliitis, ankylosing spondylitis, uveitis, skin lesions, pericholangitis, and primary sclerosing cholangitis.

Morphology

Always involves the rectum

Extends proximally in continuous pattern.

Pan colitis.

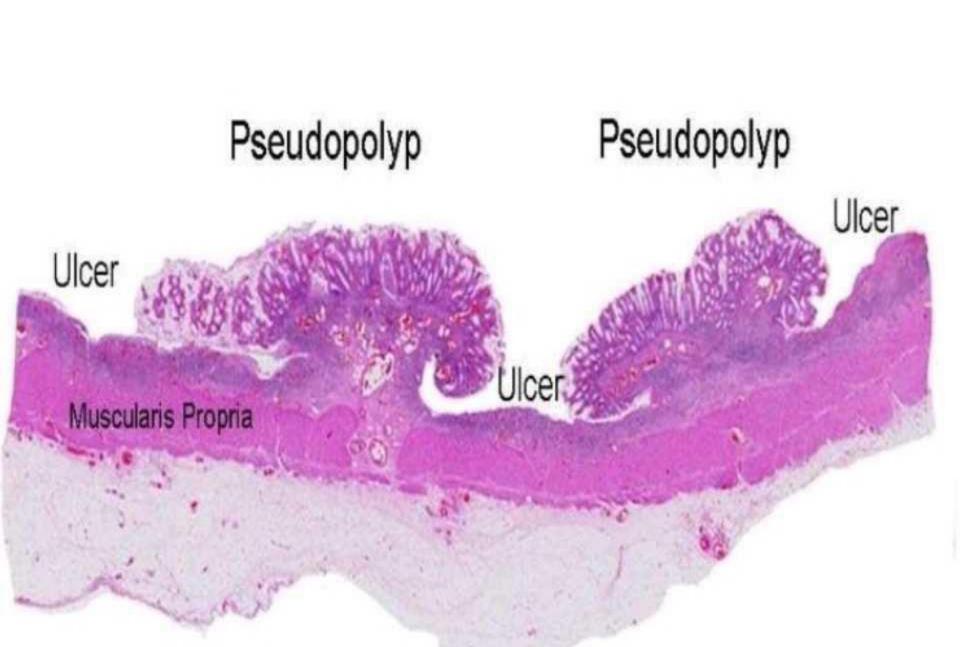
Occasionally focal appendiceal or cecal inflammation.

□ Ulcerative proctitis or ulcerative proctosigmoiditis

Small intestine is normal (except in backwash ileitis)

Macroscopic:

- Broad-based ulcers.
- Pseudopolyps
- □ Mucosal atrophy in long standing
- □ Mural thickening absent
- □ Serosal surface normal
- **No strictures**
- □ Toxic megacolon



Microscopic:

- Inflammatory infiltrates
- Crypt abscesses
- Crypt distortion
- Epithelial metaplasia
- **Submucosal fibrosis**
- Inflammation limited to mucosa and submucosa.
- **No skip lesions**
- **No granulomas.**

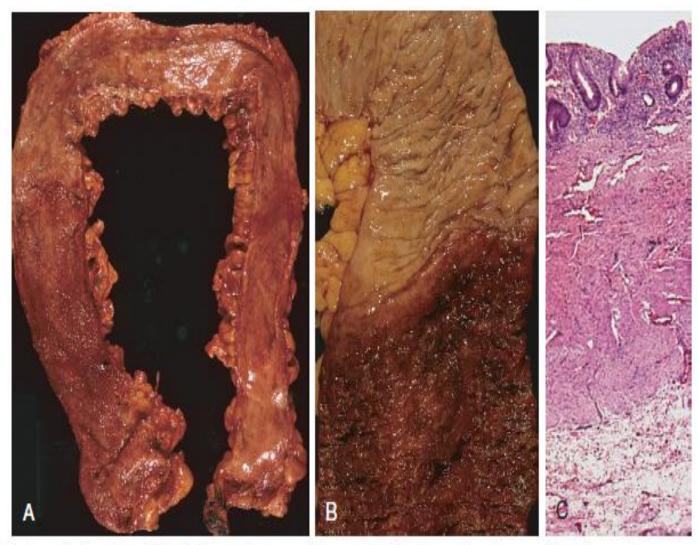
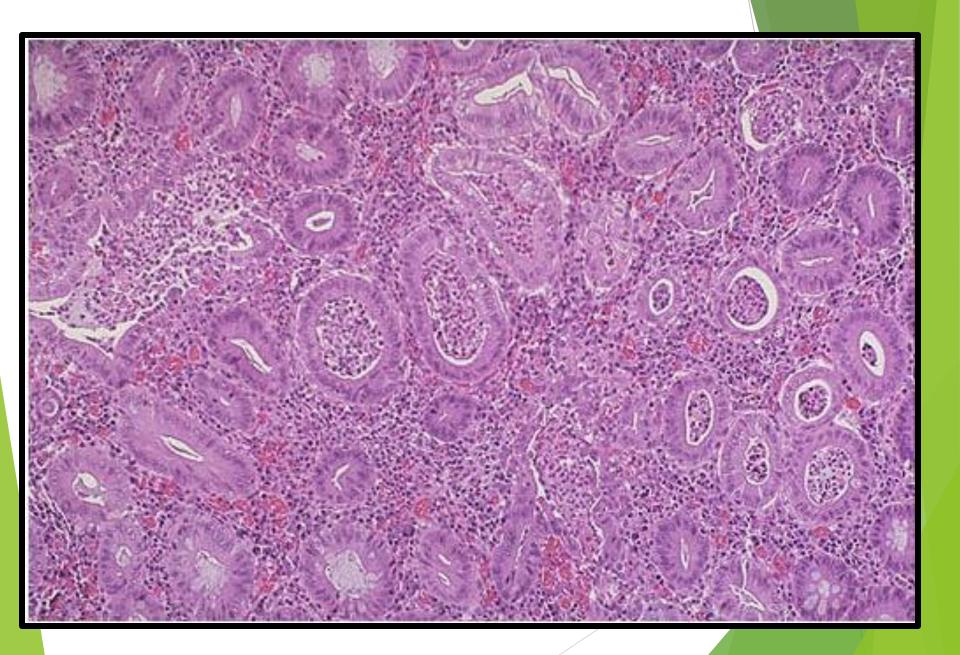


Figure 14-30 Pathology of ulcerative colitis. A, Total colectomy with pancolitis showing active disease, with red, granular mucosa in the cecum (*left*) and smooth, atrophic mucosa distally (*right*). B, Sharp demarcation between active ulcerative colitis (*bottom*) and normal (*top*). C, This full-thickness histologic section shows that disease is limited to the mucosa. Compare with Figure 14-28, C.



Clinical Features

J>Ulcerative colitis is a relapsing disorder characterized by attacks of bloody diarrhea with expulsion of stringy, mucoid material and lower abdominal pain and cramps that are temporarily relieved by defecation.

These symptoms may persist for days, weeks, or months before they Subside.

J>More than half of the patients have mild disease.

The factors that trigger ulcerative colitis are not known, but as noted previously, infectious enteritis precedes disease onset in some cases.

The initial onset of symptoms also has been reported to occur shortly after smoking cessation in some patients, and smoking may partially relieve symptoms.

Table 14-5 Features That Differ Between Crohn Disease and Ulcerative Colitis

Feature	Crohn Disease	Ulcerative Colitis
Macroscopic		
Bowel region affected	lleum ± colon	Colon only
Rectal involvement	Sometimes	Always
Distribution	Skip lesions	Diffuse
Stricture	Yes	Rare
Bowel wall appearance	Thick	Thin
Inflammation	Transmural	Limited to mucosa and submucosa
Pseudopolyps	Moderate	Marked
Ulcers	Deep, knifelike	Superficial, broad-based
Lymphoid reaction	Marked	Moderate
Fibrosis	Marked	Mild to none
Serositis	Marked	No
Granulomas	Yes (~35%)	No
Fistulas/sinuses	Yes	No
Clinical		
Perianal fistula	Yes (in colonic disease)	No
Fat/vitamin malabsorption	Yes	No
Malignant potential	With colonic involvement	Yes
Recurrence after surgery	Common	No
Toxic megacolon	No	Yes
NOTE: Not all features may be present in a single case.		

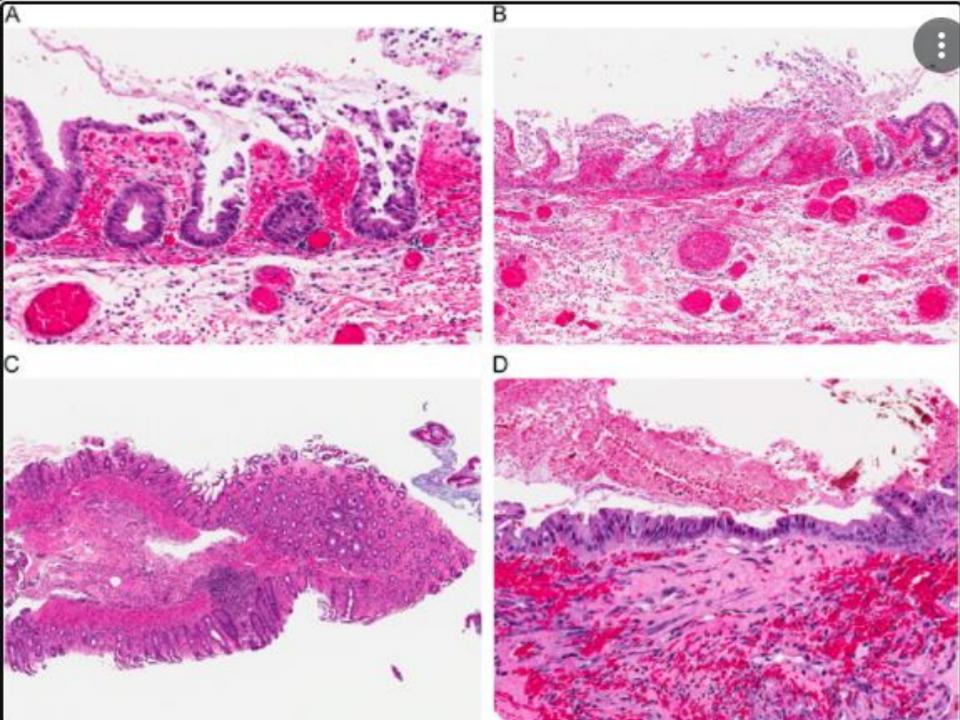
NOTE: Not all features may be present in a single case.

Ischemic Bowel Disease

Ischemic damage to the bowel wall can range from :

J> Mucosal infarction, extending no deeper than the muscularis mucosa.

- *I* **Mural infarction of mucosa and submucosa.**
- Transmural infarction involving all three layers of the wall.
- J>While mucosal or mural infarctions often are secondary to acute or chronic hypoperfusion, transmural infarction is generally caused by acute vascular obstruction.





Clinical Features

- Ischemic bowel disease tends to occur in older persons with coexisting cardiac or vascular disease.
- Acute transmural infarction typically manifests with sudden, severe abdominal pain and tenderness, sometimes accompanied by nausea, vomiting, bloody diarrhea, or grossly melanotic stool.
- Peristaltic sounds diminish or disappear, and muscular spasm creates board like rigidity of the abdominal wall.
- Mucosal and mural infarctions by themselves may not be fatal. However, these may progress to more extensive, in transmural infarction.

OTHER CAUSES

- CMV infection.
- Radiation enterocolitis .
- Necrotizing enterocolitis .
- *Angiodysplasia* is characterized by malformed submucosal and mucosal blood vessels.

The End

Good Luck