Diversion colitis: Clinical manifestations and diagnosis

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Literature review current through: **Sep 2023.**
This topic last updated: **Feb 15, 2023.**

INTRODUCTION

Diversion colitis is a nonspecific inflammatory disorder that occurs in segments of the colon and rectum that are diverted from the fecal stream by surgery (eg, creation of a loop colostomy/ileostomy or an end colostomy/ileostomy with closure of the distal colon segment [eg, Hartmann's procedure]).

Diversion colitis is characterized by inflammation of the defunctionalized, bypassed colon following surgery [1,2]. Most patients with diversion colitis are asymptomatic, but in a small proportion of patients, symptoms can significantly impact quality of life [3].

This topic will review the epidemiology, pathogenesis, clinical manifestations, and diagnosis of diversion colitis. The epidemiology, clinical manifestations, diagnosis, and management of ulcerative colitis and Crohn disease are discussed in detail separately. (See "Definitions, epidemiology, and risk factors for inflammatory bowel disease" and "Medical management of low-risk adult patients with mild to moderate ulcerative colitis" and "Management of the hospitalized adult patient with severe ulcerative colitis" and "Management of moderate to severe ulcerative colitis in adults" and "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults" and "Overview of the medical management of mild (low risk) Crohn disease in adults" and "Medical management of moderate to severe Crohn disease in adults".)

EPIDEMIOLOGY

Incidence — The true incidence of diversion colitis is unknown. Several small observational studies have demonstrated histologic changes of diversion colitis in the distal colonic segment of 70 to 100 percent of patients with fecal diversion and endoscopic evidence in 70 to 91 percent of patients [4-12].

Risk factors — Diversion colitis and diversion proctitis may develop in patients who have undergone diversion for disorders other than IBD but appear to be more common in patients with underlying inflammatory bowel disease (IBD; ie, Crohn disease and ulcerative colitis). In a study of 36 children with Hirschsprung disease, only one child (3 percent) with an ileostomy for six years developed diversion colitis [7]. In a series of 73 patients with a diverted colorectum, 87 percent of those with ulcerative colitis had symptoms compared with 30 percent of those with Crohn disease and 28 percent of those without IBD [13]. In a retrospective pathology-registry-based study of 191 patients with IBD who had a diverted rectum for more than one year, 161 (88.5 percent) had endoscopic mucosal inflammation. A total of 115 (71.4 percent) had diversion colitis, and 12 (7.5 percent) had a combination of diversion colitis and IBD in the diverted rectum [12].

PATHOGENESIS

The construction of ileostomy or colostomy with fecal diversion is routinely performed in patients who have medically refractory benign or malignant colorectal diseases, those who undergo distal bowel resection and anastomosis, or reconstruction in the distal bowel. Diversion of the fecal stream results in a deficiency of short-chain fatty acids (SCFAs) and other luminal nutrients in colonocytes in the diverted segment of the colon. It is hypothesized that the lack of these compounds or interference with their metabolism by alterations in gut flora may have a role in the development of colitis [14,15].

SCFAs, predominantly acetate, propionate, and n-butyrate, are derived from anaerobic bacterial metabolism of unabsorbed dietary carbohydrates. They are absorbed from the lumen by a combination of simple diffusion and ion exchange and oxidized by colonocytes [16]. In addition to supplying 70 percent of the fuel used by mucosal cells, luminal SCFAs have a number of other effects, including modulation of fluid and electrolyte transport, colonic motility, mucosal blood flow, and production of inflammatory cytokines [17,18]. There is a significant decrease in SCFAs and acetic acids in the luminal specimens from the diverted colon compared with those from the colostomy [19]. Other luminal nutrients for colonocytes, such as glutamine, may also play a role in the pathogenesis of diversion colitis.

Detailed bacteriologic studies have also demonstrated that the diverted segment has fewer anaerobic flora and a higher percentage of nitrate-reducing strains than controls, which may in turn result in a lack of fermentation metabolites of carbohydrates and proteins [20-22]. An increase in *Actinomyces, Anaerococcus, Corynebacterium, Peptoniphilus,* and *Porphyromonas*, and a decrease in *Lactobacillus* and *Granulicatella* have been noted in the intestinal specimens from the diverted colon compared with specimens from the colostomy [19]. However, no specific pathogens have been identified. In addition, the inconsistent response of certain patients to SCFAs in some studies and the response to restoration of fecal continuity suggest that other factors are involved in the pathogenesis of diversion colitis [23].

Mucosal immunity may play a role in the disease process of diversion colitis, although data to support this hypothesis are scant. In one study, the concentration of luminal immunoglobulin A (IgA) in the diverted colon was shown to be higher in the diverted colon than that in colostomy [19]. The results suggest that the interaction between luminal microbiota and their products and mucosal immunity is complicated.

DISEASE SPECTRUM

Diversion colitis — Diversion colitis typically occurs in diverted segments of the colon following surgery for congenital, inflammatory, or neoplastic disorders. Patients usually have a loop colostomy (or ileostomy) or an end colostomy (or ileostomy) with closure of the distal colon segment (eg, Hartmann's procedure).

Diversion pouchitis — Diversion pouchitis is a variant of diversion colitis that develops following an ileal pouch-anal anastomosis (IPAA; eg, patients with ulcerative colitis or familial adenomatous polyposis) (picture 1) [24]. The first stage of the IPAA procedure often consists of total proctocolectomy, IPAA, and a diverting loop ileostomy, which is followed by a second-stage procedure (ie, loop ileostomy closure three to six months later) [25,26]. The first stage of surgery results in temporary diversion of the fecal stream from the ileal pouch reservoir, which can lead to mucosal inflammation. In some patients with an ileal pouch, diversion may be permanent (eg, if they develop pouch failure due to refractory pouchitis or Crohn pouchitis), predisposing these patients to diversion pouchitis [27,28].

Other rare variants

- **De novo diversion-associated collagenous ileitis** – Collagenous ileitis has been reported in the efferent limb of loop ileostomy for rectosigmoid cancer [29].

- **Diversion neovaginitis** – In patients who have undergone a sigmoid vaginoplasty, diversion neovaginitis may occur [30,31].

CLINICAL MANIFESTATIONS

Signs and symptoms — Fewer than 50 percent of patients with histologic evidence of diversion colitis have symptoms, but symptoms can significantly impact quality of life [3-11]. In patients who are symptomatic, the onset usually occurs 3 to 36 months after colonic diversion [32]. The most common symptoms in adults are tenesmus, urgency, bloody and/or mucus discharge, abdominal pain, and pelvic pain or pressure [12,33]. Symptoms are mild in up to 50 percent of patients [3,11]. In rare cases, patients have severe bleeding that requires transfusion, diarrhea, or sepsis from deep ulceration and marked mucosal inflammation [11,34-37]. Patients with diversion neovaginitis present with neovaginal discharge and malodor.

Complications

Stricture — Persistent inflammation and long-term non-use may result in mild to severe stricture of the diverted bowel. Accumulation of mucus materials may cause the formation of a bezoar. The stricture can cause symptoms of abdominal or pelvic pain and discomfort, and sensation of incomplete evacuation (picture 2). In the study of 191 inflammatory bowel disease (IBD) patients with ostomies for more than one year, 57 (29.8 percent) were found to have strictures in the diverted rectum [12]. The stricture can also occur in the diverted ileal pouch (picture 2). In addition, accumulation of mucus plugs may cause the formation of a bezoar. In one study, the cumulative frequency of diversion-associated strictures in patients with underlying IBD was 29 percent [12].

Stricture at diverted bowel can be short or long with a cut-off of 4 to 5 cm (image 1). The most common locations are the distal rectum, pouch, anus, or anastomosis (picture 3). Purely diversion-associated stricture often results in web-like strictures. The stricture can be inflammatory, fibrotic, or both. Fibrotic strictures mainly occur in the IPAA, ileorectal anastomosis, or colorectal anastomosis in patients with diverting ostomies. In severe cases, the outlet of diverted bowel can be completely sealed (image 1) [38]. Long-term fecal diversion may also result in stricture formation in the distal bowel.

Inflammatory bowel disease — Inflammation in the diverted segment of the large bowel may also set the stage for the development of symptomatic "ulcerative colitis"-like condition in the in-stream colon proximal to the diverted segment [39-42]. This has been illustrated in case reports of patients who had undergone sigmoid colostomies who developed diversion colitis

[39]. Neither had a family history of IBD, but both patients later developed bleeding through the colostomy, with endoscopic and histologic features of ulcerative colitis in the previously normal colon proximal to the colostomy. Studies suggest that both innate and adaptive immunities of diverted colon mucosa are altered [43,44]. A plausible hypothesis for the pathogenesis of proximal colitis is that leukocytes sensitized and activated in the vasculature of the diverted segment are recruited to the genetically susceptible proximal mucosa.

Colorectal risk based on underlying risk factors — The risk of dysplasia or cancer in the diverted colorectum appears to be low in patients with diversion colitis in the absence of a history of colorectal cancer (CRC) or IBD [45,46]. Surveillance for CRC in the diverted segment is not required unless the underlying condition for which the surgery was performed is associated with an increased risk of CRC (eg, preoperative diagnosis of CRC or a long history of IBD involving the colon and rectum) [12,47-49]. Surveillance biopsy of friable mucosa in the diverted bowel carries a higher risk for procedure-associated bleeding than nondiverted bowel. In addition, diagnosis of dysplasia with diversion-associated background inflammation can be challenging [50]. (See "Post-treatment surveillance after colorectal cancer treatment" and "Surveillance and management of dysplasia in patients with inflammatory bowel disease".)

DIAGNOSIS

Diversion colitis should be suspected in patients with defunctionalized, diverted distal colon (with or without preexisting inflammatory bowel disease (IBD)) with cramping abdominal pain and a mucoid or bloody discharge. As the clinical manifestations, endoscopic and histologic findings of diversion colitis are nonspecific, the diagnosis requires evaluation to exclude other etiologies. (See 'Endoscopy with biopsy' below and 'Differential diagnosis' below.)

EVALUATION

Goals of the diagnostic evaluation for patients with suspected diversion colitis are to exclude other causes of symptoms, establish the diagnosis of diversion colitis, and determine the severity of the disease. (See 'Differential diagnosis' below.)

History — The history serves to identify symptoms that are suggestive of other conditions. Risk factors for other causes of colitis should be sought.

- **Systemic symptoms** – Systemic symptoms including fever, chills, sweats, and weight loss are suggestive of an infectious cause of symptoms (eg, *Clostridioides difficile*) or Crohn disease.

- **Exposures** – This includes a history of recent travel to areas endemic for bacterial or parasitic infections including amebiasis and a history of or risk factors for sexually transmitted diseases (eg, *Neisseria gonorrhea* and herpes simplex virus [HSV]) that are associated with proctitis. Travel to a resource-limited setting increases the risk of bacterial diarrhea and also informs the risk of certain parasitic infections.

- **Medical history** – It is also important to ask about nonsteroidal antiinflammatory drug (NSAID)/medication exposure, recent antibiotic use (as a clue to the presence of *C. difficile* infection), other medications (such as proton pump inhibitors, which can increase the risk of infectious diarrhea), and to obtain a complete past medical history (eg, to identify an immunocompromised host or the possibility of nosocomial infection). Atherosclerotic disease or prior ischemic episodes are suggestive of chronic colonic ischemia. A history of abdominal/pelvic radiation should be sought as these may also be associated with colitis. In an immunocompromised patient, cytomegalovirus (CMV) infection can mimic diversion colitis. (See "Clinical manifestations, diagnosis, and prognosis of ulcerative colitis in adults", section on 'Differential diagnosis' and "Radiation proctitis: Clinical manifestations, diagnosis, and management" and "NSAIDs: Adverse effects on the distal small bowel and colon" and "Colonic ischemia".)

Laboratory studies — For patients with suspected diversion colitis, we obtain the following laboratory studies:

- **Stool studies to exclude infection** – Our approach to obtaining stool studies includes:

  - Stool *C. difficile* toxin, routine stool cultures (*Salmonella*, *Shigella*, *Campylobacter, Yersinia*), and specific testing for *Escherichia coli* O157:H7.

  - Microscopy for ova and parasites (three samples) and a *Giardia* stool antigen test, particularly if the patient has risk factors such as recent travel to endemic areas.

  - Testing for sexually transmitted infections, including *C. trachomatis, N. gonorrhoeae*, HSV, and *Treponema pallidum*, particularly in men who have sex with men or patients with severe rectal symptoms including urgency and tenesmus. (See "Clinical manifestations and diagnosis of *Neisseria gonorrhoeae* infection in adults and adolescents" and "Epidemiology, clinical manifestations, and diagnosis of genital herpes simplex virus in patients with HIV" and "Syphilis: Screening and diagnostic testing".)

- **Stool inflammatory markers** – Although tests for inflammation, fecal calprotectin or lactoferrin are not used routinely to diagnose diversion colitis and do not replace

endoscopic evaluation, they may be used to assess disease severity.

- **Blood tests** – Blood tests include a complete blood count, electrolytes, albumin, and C-reactive protein (CRP).

Endoscopy with biopsy — Endoscopy and biopsy play a key role in the diagnosis of diversion colitis.

Procedure technique — The mucosa of diverted bowel is normally friable and barotrauma can occur with air insufflation even in the absence of diversion colitis (image 2). Therefore, endoscopic assessment of inflammation of the diverted large bowel or the ileal pouch is made on insertion rather than withdrawal and findings should be photo-documented. Existing endoscopic activity indices might not be reliable in this setting and should not be used [50].

We use the following techniques to minimize the risk of bleeding, bacterial translocation, and perforation:

- Endoscopy is performed using a standard gastroscope or pediatric colonoscope.
- The procedure is performed with minimal air insufflation or preferably with carbon dioxide insufflation.
- Mucosal biopsies are obtained with standard forceps and deep or jumbo biopsies are avoided [51,52].

Endoscopy findings — Endoscopic features of diversion colitis are nonspecific and include erythema, friability, edema, mucus plugs, and granularity of the colorectal mucosa. In more severe cases, patients may have ulcers, nodularity, edema, or inflammatory and filiform polyps, strictures, or fecal bezoars [53]. In some patients, endoscopy may show a "cat scratch" pattern with bright erythematous linear marks. In some patients with outlet stricture, there might be an accumulation of large quantity of exudate and mucus, which can cause bezoar formation (picture 3 and image 3).

Histopathology — Histopathologic features of diversion colitis are not diagnostic, but biopsies of the colon are necessary to exclude other causes of colitis. The most common pathologic features of diversion colitis include expanded lymphoid aggregates and inflammation in the lamina propria with lymphocytes and plasma cells (picture 4) [1,7,54] and regenerative gland and mucin-depleted glands [54]. Although crypt architecture is usually preserved, features that mimic IBD, including crypt distortion and basal lymphoplasmacytosis, may be seen [5,11,32]. In cases with marked lymphoid hyperplasia, the crypts may appear atrophic, short, and displaced by the lymphoid infiltrate, imparting a false appearance of crypt architectural abnormalities of

IBD [55]. Cryptolytic or suture-associated granulomas may be present in diversion proctocolitis. However, non-cryptolytic granulomas are rare [56].

In patients with a diverted bowel, histopathologic manifestations of architectural distortion, disease activity, mucosal atrophy, ulcers, pyloric gland metaplasia, granulomas, intramucosal lymphoid aggregates, transmural inflammation, and transmural lymphoid aggregates are all more common in those with underling IBD than those without IBD [57]. (See 'Differential diagnosis' below.)

Limited role for abdominal imaging — We do not routinely obtain imaging (eg, cross-sectional imaging, pouchogram with water-soluble contrast) for patients with suspected diversion colitis as the diverted bowel may have nonspecific thickened wall and mucosal hyperenhancement on contrasted cross-sectional imaging regardless of the presence of inflammation (picture 5). For patients with symptoms that are suggestive of a stricture, water-soluble contrasted enemas via stoma or anus may help delineate the presence and nature of strictures of the diverted bowel (image 1).

DIFFERENTIAL DIAGNOSIS

In order to diagnose diversion colitis, it is important to distinguish it from other disorders that can have similar clinical manifestations [58]. The clinical history provides important information to narrow the differential diagnosis, but additional evaluation may be needed. There are distinct and shared features on clinical, endoscopic, and histologic findings between ulcerative proctitis, chronic radiation proctopathy, and diversion proctitis (table 1).

- **Infectious colitis** – Infectious colitis may have a similar clinical presentation and endoscopic appearance to diversion colitis (table 2). Infectious colitis can be excluded with stool and tissue cultures, stool studies, and on biopsies of the colorectum. (See 'Laboratory studies' above.)

- **Inflammatory bowel disease** – In patients with distal colonic involvement of IBD prior to surgery, it is difficult to differentiate recurrent active IBD in the diverted colon from diversion colitis [59]. The documentation of the presence or absence of IBD in the diverted colon segment prior to surgery is helpful in distinguishing recurrent IBD in the diverted colon from diversion colitis. In addition, certain histologic features may be helpful (table 1):

  - The histology pattern of enterocolic lymphocytic phlebitis, characterized by artery-sparing and venulocentric lymphoid infiltration, is more commonly seen in patients

with IBD and superimposed diversion colitis as compared with those with IBD alone [60].

- The presence of noncaseating, nonmucinous granulomas in the defunctionalized rectum is suggestive of the presence of Crohn disease [61].

- The presence of a diffuse cellular infiltrate and prominent lymphoid follicular hyperplasia is suggestive of diversion colitis [7,13,32,60].

In some cases, this distinction may still not be possible. In such cases, a response to a trial of short-chain fatty acid enemas is suggestive of diversion colitis. On the other hand, a favorable response to systemic corticosteroid or anti-tumor necrosis factor (TNF) biologics in a patient with diverted colorectum suggests the dominant role of underlying IBD in symptomatology.

- **Radiation colitis/proctitis** – Endoscopic and histologic features of diversion colitis can mimic those seen in radiation colitis/proctitis. Radiation colitis may be seen weeks to years after abdominal or pelvic irradiation. Although not specific for radiation colitis/proctitis, histologic findings suggestive of radiation colitis include eosinophilic infiltrates, epithelial atypia, fibrosis, and capillary telangiectasia (table 1). (See "Radiation proctitis: Clinical manifestations, diagnosis, and management", section on 'Clinical suspicion' and "Radiation proctitis: Clinical manifestations, diagnosis, and management", section on 'Clinical manifestations'.)

- **Medication-associated colitis** – Nonsteroidal antiinflammatory drugs (NSAIDs) can cause chronic diarrhea and bleeding [62]. Other drugs associated with a similar clinical presentation include retinoic acid and gold. Immune checkpoint inhibitors, including cytotoxic T lymphocyte antigen 4 (CTLA-4) inhibitors (eg, ipilimumab), and programmed cell death protein 1 (PD-1) and programmed cell death protein ligand 1 (PD-L1) inhibitors (eg, pembrolizumab and nivolumab), are associated with colitis [63,64]. The diagnosis is established by a history of medication use and the presence of nonspecific mucosal inflammation or mucosal erosions on endoscopy and biopsy, and in some cases, resolution of symptoms and mucosal inflammation after the cessation of the culprit medications. (See "NSAIDs: Adverse effects on the distal small bowel and colon" and "Immune checkpoint inhibitor colitis".)

- **Ischemic colitis** – Patients with ischemic colitis have a history of underlying vascular disease or other risk factors (eg, aortic surgery, cardiopulmonary bypass, myocardial infarction, hemodialysis, dehydration, thrombophilia). Colonoscopic findings include pale mucosa with petechial bleeding. Bluish hemorrhagic nodules may be seen. More severe

disease is marked by cyanotic mucosa and hemorrhagic ulcerations. Ischemia colitis is segmental in distribution with abrupt transition between injured and non-injured mucosa, and rectal sparing. A single linear ulcer running along the longitudinal axis of the colon may also be suggestive of ischemic colitis [65]. Biopsies taken from affected areas may show nonspecific changes such as intra- and/or submucosal hemorrhage, crypt destruction or crypt drop-out, capillary thrombosis, granulation tissue with crypt abscesses, and pseudopolyps, which may mimic Crohn disease [66,67]. In the chronic phase of ischemic colitis, mucosal atrophy and areas of granulation tissue may be seen. Chronic ischemic colitis may manifest as colonic strictures. Biopsy of a post-ischemic stricture is marked by extensive transmural fibrosis and mucosal atrophy. The clinical presentation and diagnosis of ischemic colitis is discussed in detail separately. (See "Colonic ischemia", section on 'Clinical features'.)

SUMMARY AND RECOMMENDATIONS

- **Epidemiology** – Diversion colitis is an inflammatory process that occurs in segments of the colorectum that are diverted from the fecal stream by surgery. Histologic changes of diversion colitis may be seen in the distal colonic segment of 70 to 100 percent of patients with fecal diversion. (See 'Epidemiology' above.)

- **Disease spectrum** – Diversion colitis typically occurs in diverted segments of the colon following surgery for congenital, inflammatory, or neoplastic disorders. Patients usually have a loop colostomy (or ileostomy) or an end colostomy (or ileostomy), with closure of the distal colon segment (eg, Hartmann's procedure). Diversion pouchitis is a variant of diversion colitis that develops following an ileal pouch-anal anastomosis (IPAA; eg, patients with ulcerative colitis or familial adenomatous polyposis). (See 'Disease spectrum' above.)

- **Pathogenesis** – Diversion of the fecal stream results in a deficiency of short-chain fatty acids (SCFAs), along with other luminal nutrients needed by colonocytes in the diverted segment. It is hypothesized that the lack of these compounds or interference with their metabolism by alterations in gut flora may have a role in the development of colitis. However, the inconsistent response of patients to SCFAs and the almost uniform response to restoration of fecal continuity suggest that other factors are involved in the pathogenesis of diversion colitis. (See 'Pathogenesis' above.)

- **Clinical manifestations** – Fewer than 50 percent of patients with histologic evidence of diversion colitis have symptoms, but symptoms can significantly impact quality of life. The

most common symptoms in adults are tenesmus, urgency, bloody and/or mucus discharge, and abdominal pain. Symptoms are mild in up to 50 percent of patients. In rare cases, patients have severe bleeding that requires blood transfusion, diarrhea, or sepsis from deep ulceration. (See 'Clinical manifestations' above.)

- **Complications** – Persistent inflammation and long-term non-use may result in mild to severe stricture of the diverted bowel, especially in the diverted ileal pouch. In addition, accumulation of mucus materials may cause the formation of a bezoar. The stricture can cause symptoms of abdominal or pelvic pain and discomfort, and a sensation of incomplete evacuation. (See 'Complications' above.)

- **Diagnostic evaluation** – Diversion colitis should be considered in an individual with or without preexisting inflammatory bowel disease who complains of cramping abdominal pain with a mucoid or bloody discharge from the defunctionalized, bypassed distal colon. Since the clinical manifestations of diversion colitis are nonspecific, the diagnosis requires exclusion of other disorders by history, laboratory studies to rule out infection (table 2), and endoscopy with biopsy (table 1). (See 'Evaluation' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Dr. Richard I. Breuer, who contributed to an earlier version of this topic review.

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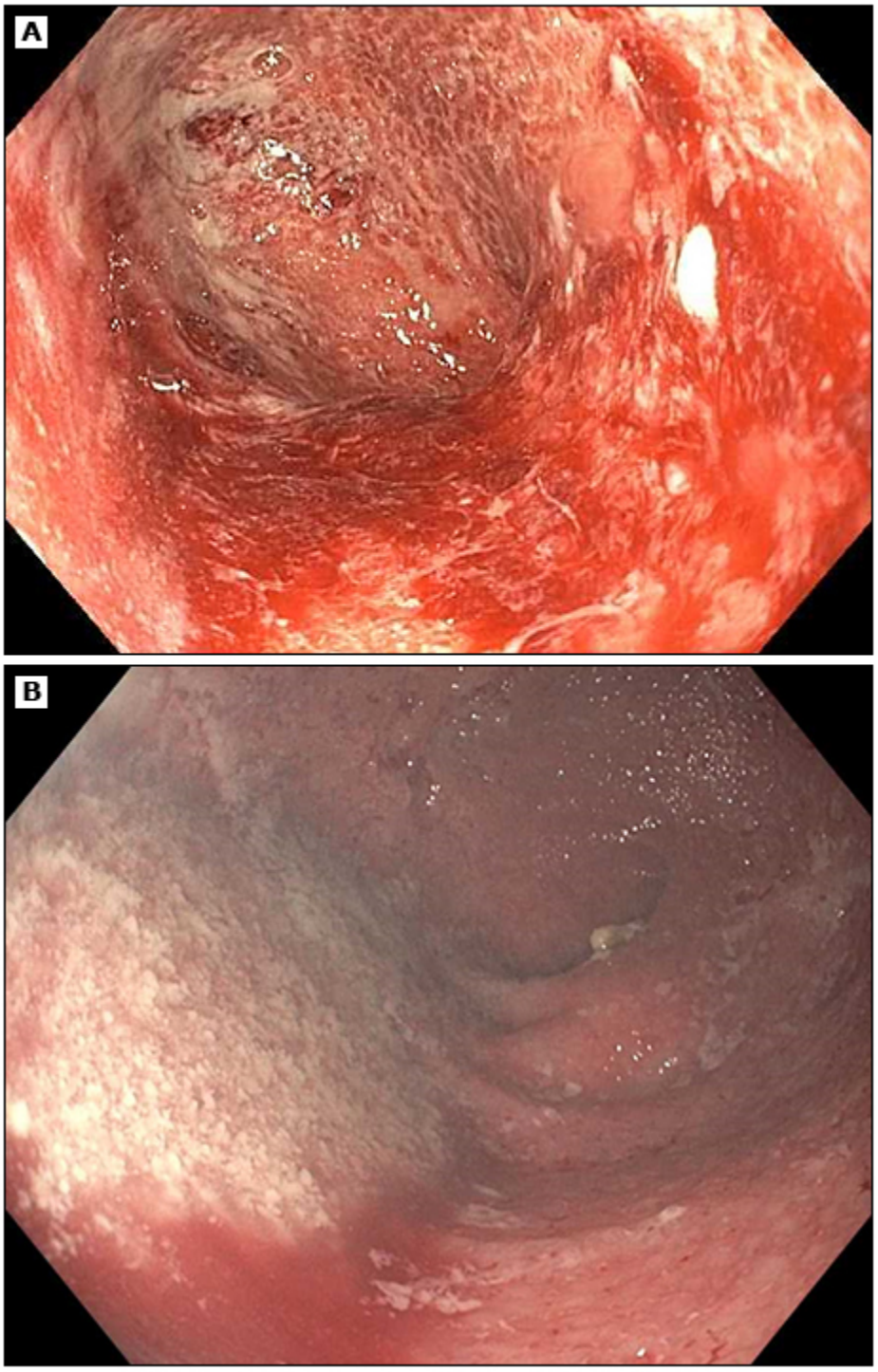
Topic 4075 Version 22.0

GRAPHICS

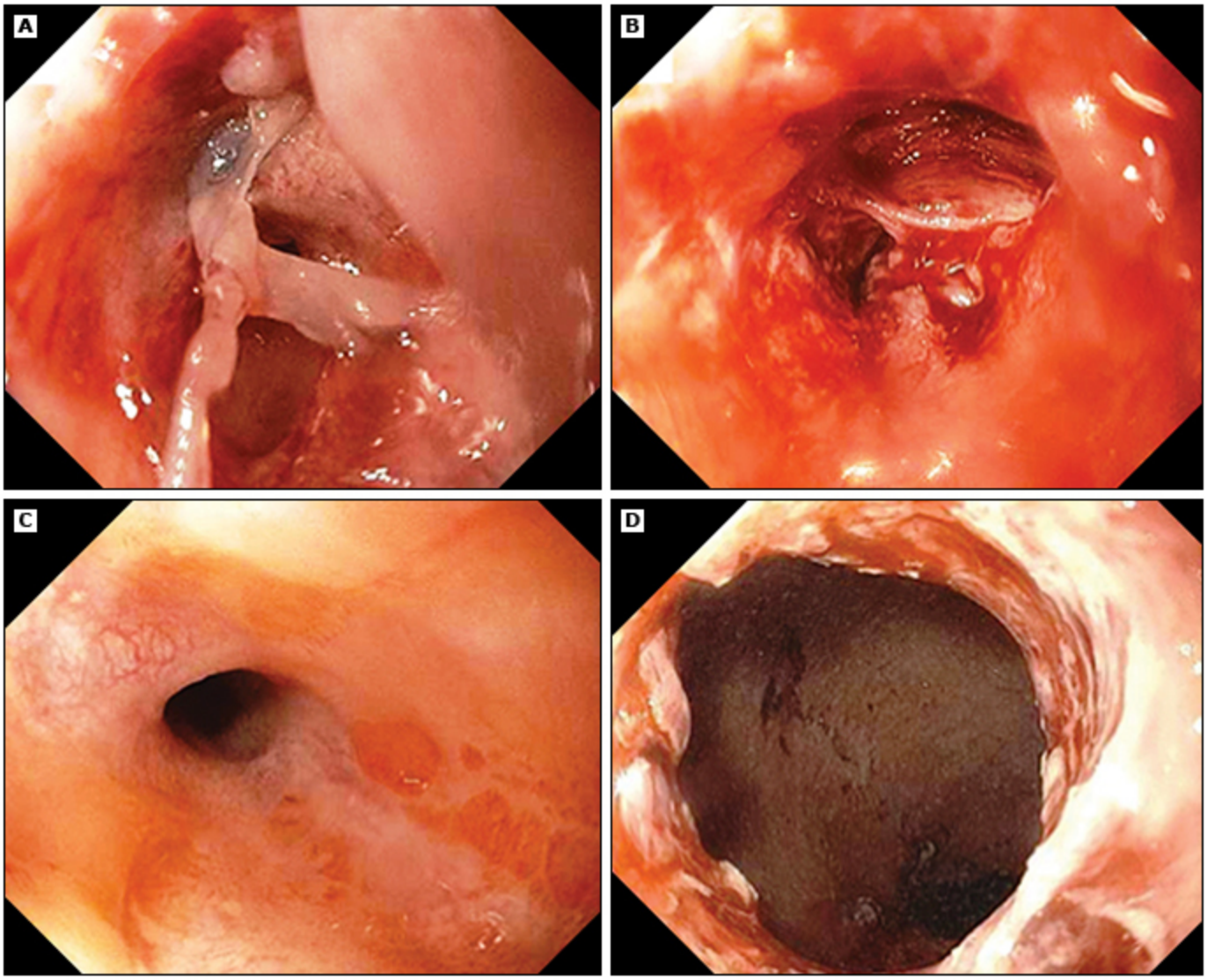
Diversion pouchitis



Endoscopic images of diversion pouchitis with severe edema, friability, and hemorrhage of the pouch mucosa.

Graphic 90181 Version 2.0

Stricture complicating diversion colitis



Tight stricture in the diverted bowel.

(A) Tight anastomotic stricture prior to balloon dilation.

(B) Post-balloon dilatation with trauma.

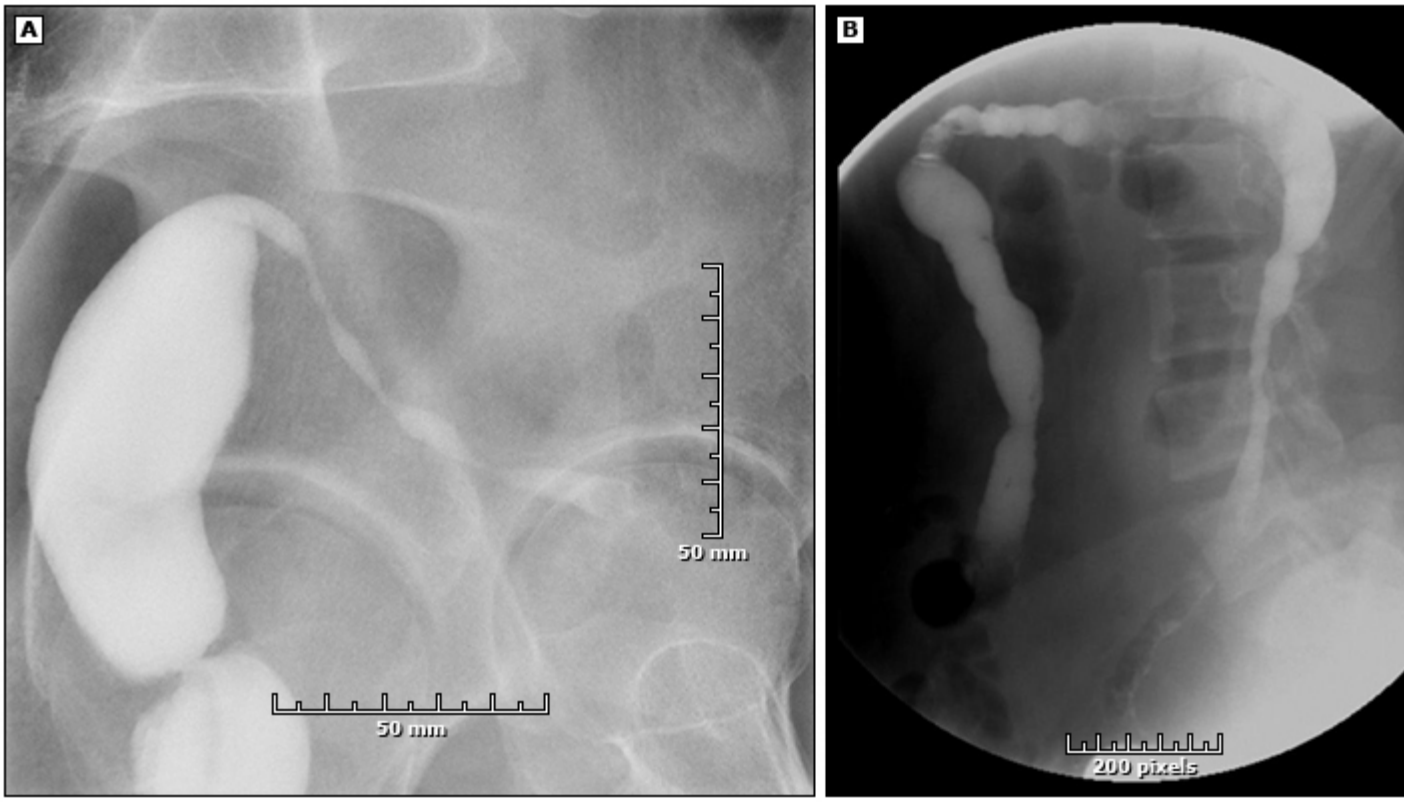
(C) Tight anastomotic stricture prior to needle-knife stricturotomy.

(D) Post-needle-knife stricturotomy.

From: Sinh P, Shen B. Endoscopic evaluation of surgically altered bowel in patients with inflammatory bowel diseases. Inflamm Bowel Dis 2015; 21:1459. DOI: 10.1097/MIB.0000000000000357.

Graphic 101840 Version 2.0

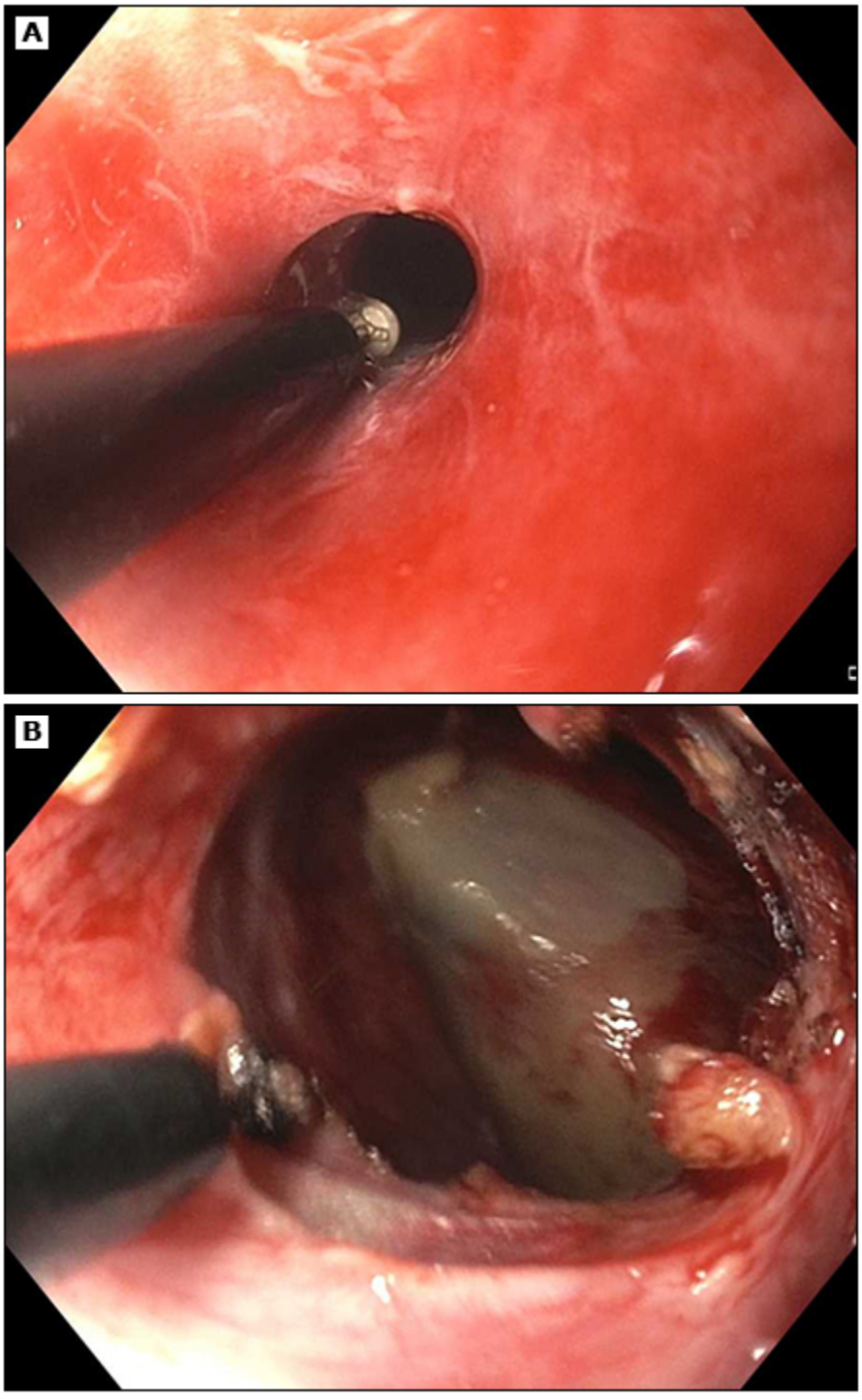
Long strictures of diverted colon



Long strictures of diverted colon (A, B).

Graphic 118677 Version 1.0

Diversion-associated stricture



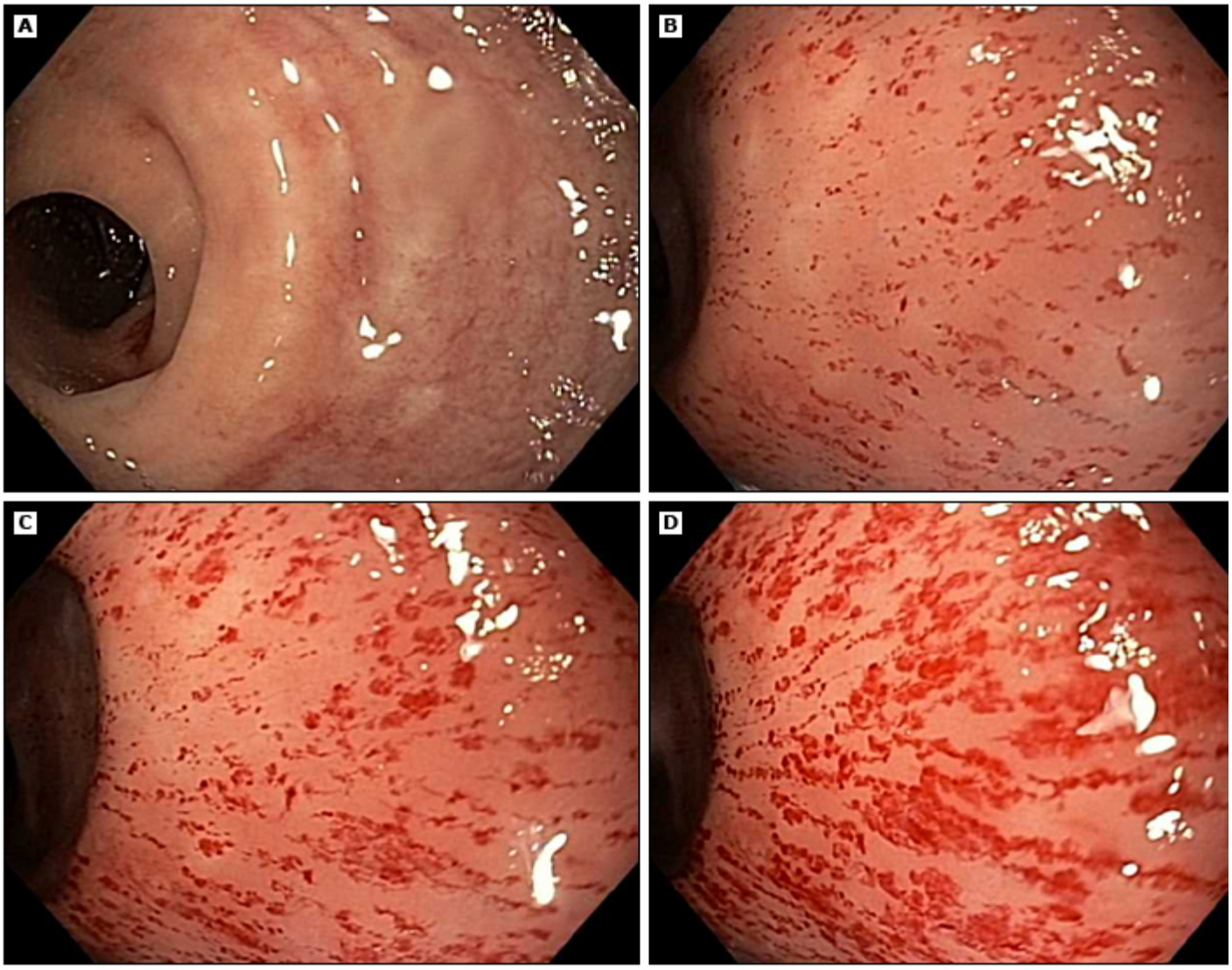
Diverted large bowel in a patient with Crohn disease.

(A) Diversion-associated anorectal stricture.

(B) Endoscopic stricturotomy of a diversion-associated stricture.

Graphic 139433 Version 1.0

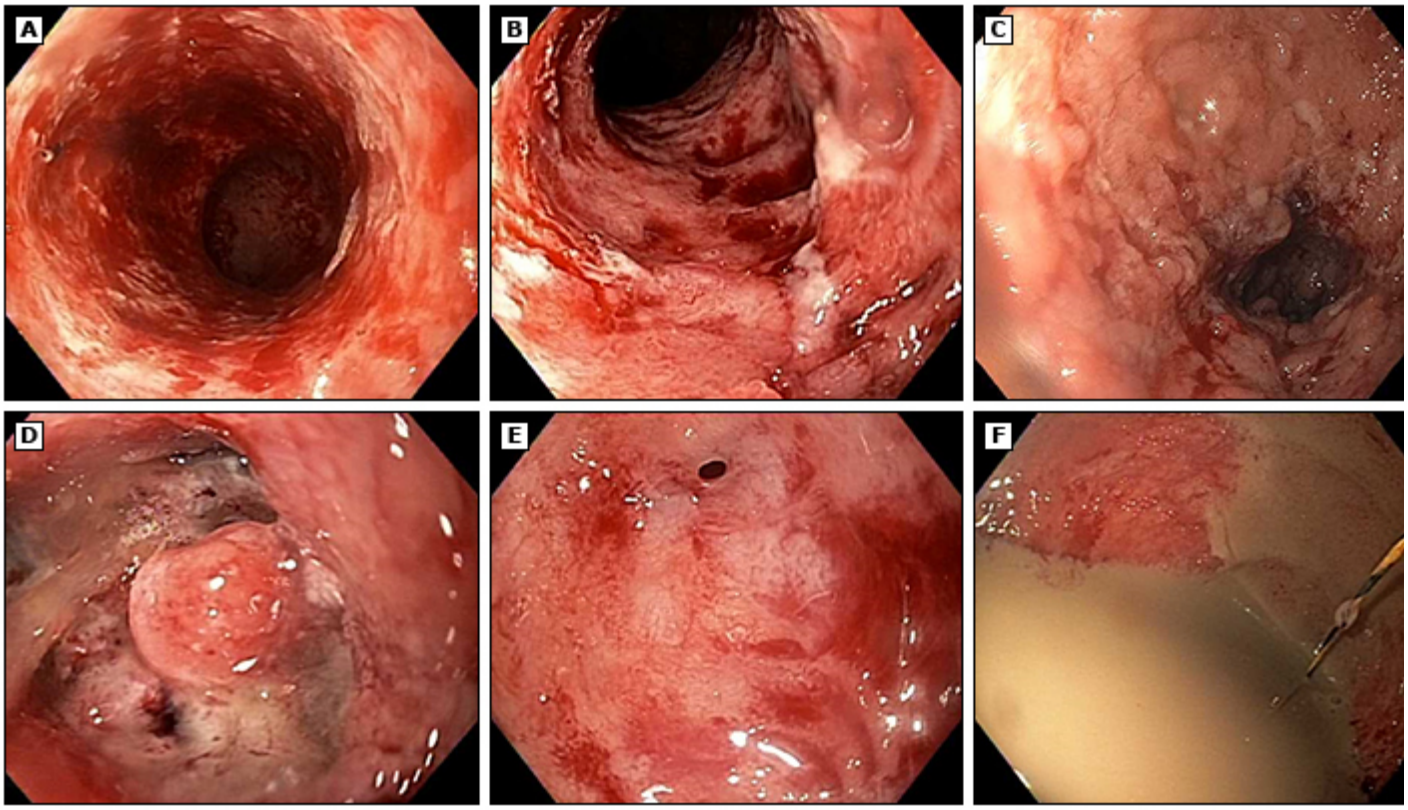
Diverted colon with edematous mucosa



Diverted colon. Slight edematous mucosa (A) becomes friable even with gentle air insufflation during endoscopy (B-D). The feature is almost uniformly present in all patients with diverted bowel to a certain degree.

Graphic 118675 Version 2.0

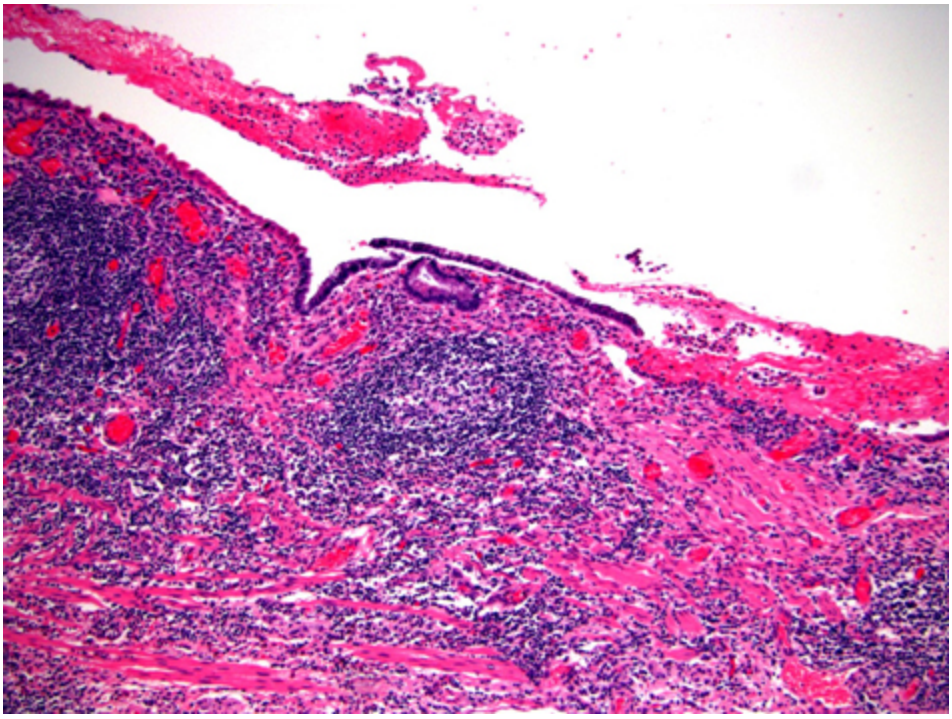
Pattern of disease of diverted bowel



Spectrum of Diversion Colitis. Diffuse inflammation with friable mucosa (A, B), nodularity (C), inflammatory polyps (D), severe outlet stricture (E), and copious exudates in the lumen (F).

Graphic 118676 Version 1.0

Diversion colitis histology



Histology of diversion colitis characterized by reactive lymphoid hyperplasia, as well as crypt atrophy.

From: Wu XR, Liu XL, Katz S, Shen B. Pathogenesis, diagnosis, and management of ulcerative proctitis, chronic radiation proctopathy, and diversion proctitis. Inflamm Bowel Dis 2015; 21:703. DOI: 10.1097/MIB.0000000000000227. Reproduced with permission from Lippincott Williams & Wilkins. Copyright © 2015 Crohn's and Colitis Foundation of America, Inc. Unauthorized reproduction of this material is prohibited.

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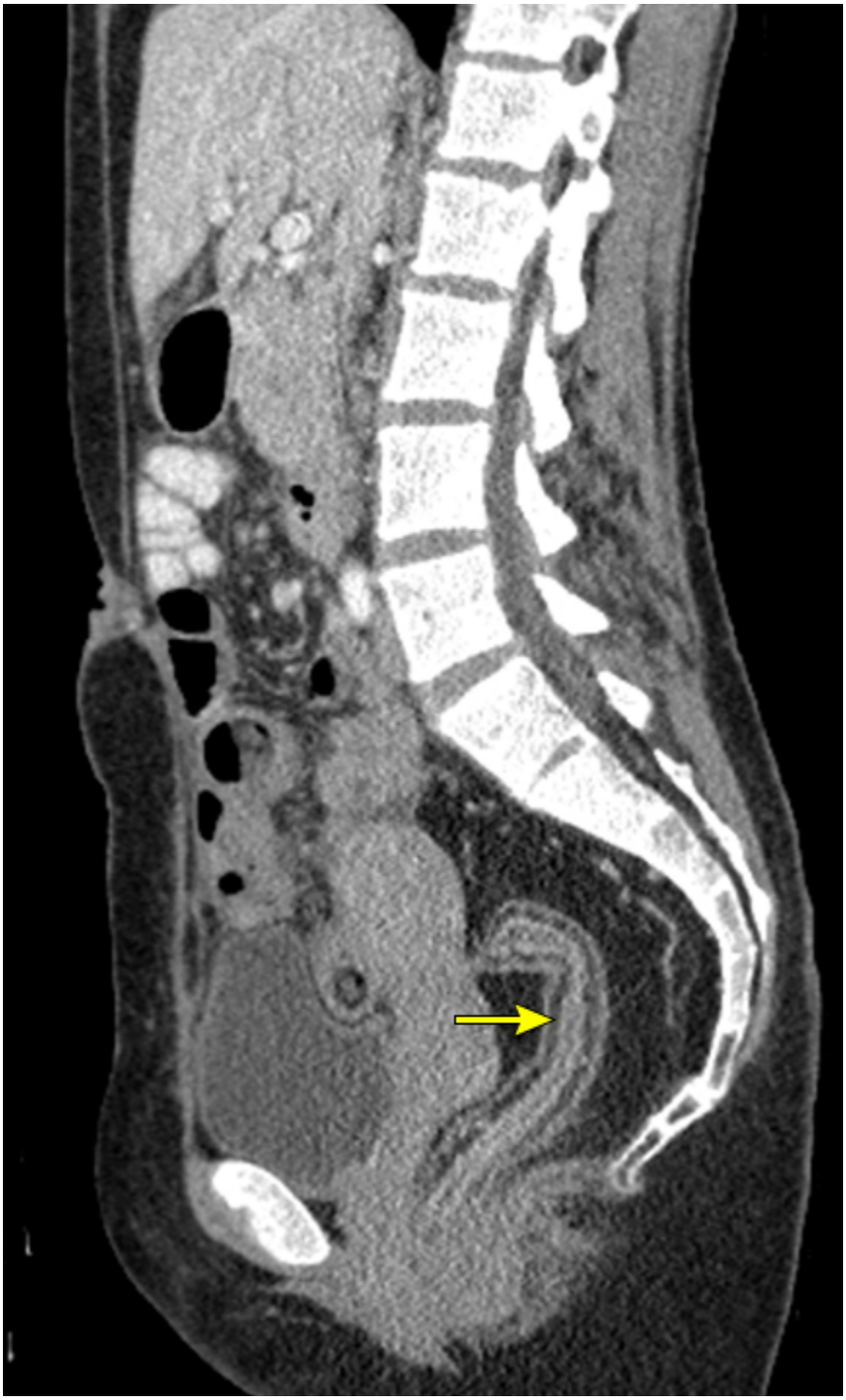
Bowel wall edema in diversion colitis



Bowel wall edema and mucosal hyperenhancement in diversion colitis on computed tomography.

Graphic 139434 Version 1.0

Comparison of ulcerative proctitis, chronic radiation proctopathy, and diversion proctitis

Characteristics	Ulcerative proctitis	Chronic radiation proctopathy	Diversion proctitis
Shared clinical features	Rectal bleeding, diarrhea, urgency, tenesmus, incontinence, pelvic pain, sense of incomplete evacuation		
Disease-specific clinical features	Risk of proximal extension of inflammation; ulcerative proctitis improves after fecal diversion	Excessive bleeding; inflammation persists even after fecal diversion	Bloody mucus discharge; inflammation resolves after reestablishment of continuity
Shared endoscopic features	Edema, erythema, friability, ulcer, exudates		
Disease-specific endoscopic features	Sharp demarcation of diseased and non-diseased segments of distal large bowel; inflammatory polyps	Angiectasia or arteriovenous malformation-like lesions	Anal or distal rectal stricture, bleeding induced by endoscopic air insufflation; "cat scratch"
Shared histologic features	No definite shared histologic features		
Disease-specific histologic features	Prominent basal lymphoplasmacytosis	Dilated, tortuous capillaries, prominent endothelial cell nuclei, surrounded by a cuff of hyalinized lamina propria	Follicular lymphoid hyperplasia
Other histologic features	Epithelial injury from mucin depletion, cryptitis, crypt abscess, erosion and ulceration; crypt distortion; Paneth cell metaplasia; hypertrophy of muscularis mucosae in some cases	Variable degree of epithelial injury, crypt distortion, erosion and ulceration; sclerosis of submucosal arteries, the presence of atypical stellate fibroblasts, and secondary chronic ischemic changes such as crypt atrophy and Paneth cell metaplasia	Diffuse mild acute inflammation, lymphoplasmacytic infiltrate most dense in the superficial epithelia; may show mucin depletion, cryptitis, crypt abscess, crypt atrophy, and Paneth cell metaplasia

Modified from: Wu XR, Liu XL, Katz S, Shen B. Pathogenesis, diagnosis, and management of ulcerative proctitis, chronic radiation proctopathy, and diversion proctitis. Inflamm Bowel Dis 2015; 21:703. DOI: 10.1097/MIB.0000000000000227.

Graphic 101839 Version 5.0

Infectious mimics of inflammatory bowel disease

Infectious agents causing colitis and/or ileitis
Bacteria
Shigella species
Enterohemorrhagic *Escherichia coli*
Enteroinvasive *E. coli*
Campylobacter jejuni
Salmonella species (gastroenteritis and typhoid fever)
Yersinia enterocolitica
Mycobacterium tuberculosis
Clostridioides (formerly *Clostridium*) *difficile*
Vibrio parahaemolyticus
Chlamydia trachomatis (lymphogranuloma venereum serotypes)
Parasites
Entamoeba histolytica
Schistosoma species
Balantidium coli
Trichinella spiralis
Viruses
Cytomegalovirus
Infectious agents causing proctitis
Neisseria gonorrhoeae
Herpes simplex virus
C. trachomatis
Treponema pallidum
Cytomegalovirus

Adapted from: Guerrant RL, Lima AA. Inflammtory enteritides. In: Principles and Practice of Infectious Diseases, 5th ed, Mandell GL, Bennett JE, Dolin R (Eds), Churchill Livingstone, Philadelphia 2000. p.1127.

Graphic 67648 Version 4.0

Contributor Disclosures

Bo Shen, MD, AGAF, FACG, FASGE Grant/Research/Clinical Trial Support: Takeda [Crohn disease and Pouchitis]. Consultant/Advisory Boards: Abbvie [Crohn disease, ulcerative colitis]; Janssen [Crohn disease, ulcerative colitis]. All of the relevant financial relationships listed have been mitigated. **J Thomas Lamont, MD** Equity Ownership/Stock Options: Allurion [Weight loss]. Consultant/Advisory Boards: Teledoc [Gastrointestinal diseases]. All of the relevant financial relationships listed have been mitigated. **Shilpa Grover, MD, MPH, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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