Pouchitis: Epidemiology, pathogenesis, clinical features, and diagnosis

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INTRODUCTION

Restorative proctocolectomy with ileal pouch-anal anastomosis (IPAA) is often performed for patients with ulcerative colitis (UC) and familial adenomatous polyposis (FAP) who require colectomy. However, patients with IPAA are at risk of pouch-related disorders (eg, inflammatory, postsurgical). Pouchitis is an inflammatory disorder that typically presents with increased stool frequency and urgency and is a common complication of IPAA or a continent ileostomy (eg, Kock pouch).

This topic will review the epidemiology, pathogenesis, clinical features, and diagnosis of acute pouchitis. Medical management of acute and chronic pouchitis is discussed separately. (See "Management of acute and chronic pouchitis".)

Surgical management of UC is discussed separately. (See "Surgical management of ulcerative colitis".)

Management of FAP is discussed separately. (See "Familial adenomatous polyposis: Screening and management of patients and families".)

Postoperative complications and functional results of proctocolectomy with ileal pouch-anal anastomosis are discussed separately. (See "Restorative proctocolectomy with ileal pouch-anal anastomosis: Laparoscopic approach".)

The International Ileal Pouch Consortium has published guidelines on the diagnosis and classification of ileal pouch disorders, and our approach is consistent with those guidelines [1].

EPIDEMIOLOGY

Incidence — Among patients who have undergone an ileal pouch-anal anastomosis (IPAA), the reported incidence of pouchitis ranges from 20 to 50 percent [2-6]. Pouchitis occurs more frequently among patients who have undergone an IPAA for ulcerative colitis than those with familial adenomatous polyposis [2,7]. (See "Clinical manifestations and diagnosis of familial adenomatous polyposis".)

Age — Patients may present with acute pouchitis at any age.

SPECTRUM OF DISEASE

Many patients with ileal pouches have some degree of endoscopic inflammation of the ileal pouch reservoir, and pouchitis likely represents a disease spectrum with a range of presentations that may evolve over time. In addition, disease characteristics (eg, response to antibiotics, frequency of relapses) have been used to classify pouchitis and help guide treatment. As an example, acute antibiotic-responsive pouchitis may evolve into chronic antibiotic-refractory pouchitis. (See 'Etiopathogenesis' below and "Management of acute and chronic pouchitis".)

Pouchitis and other ileal pouch disorders have been characterized based on the following factors [1,8,9]:

- Duration of symptoms – Acute (≤4 weeks) versus chronic (>4 weeks).
- Response to antibiotics – Antibiotic-responsive versus antibiotic-dependent (ie, requiring ongoing antibiotic therapy to keep disease in remission) versus antibiotic-refractory (ie, not responding to ≥4 weeks of antibiotic therapy) [10].
- Frequency of flares – Episodic pouchitis (<3 episodes per year) versus relapsing pouchitis (≥3 episodes per year) versus chronic pouchitis.
- Disease activity – Active inflammation versus disease in remission.
- Etiology – For most patients, the cause of pouchitis is uncertain but possibly related to intestinal microbiota (ie, dysbiosis of commensal bacteria). (See 'Etiopathogenesis' below.)

However, for some patients, pouchitis is associated with a specific etiology (ie, secondary pouchitis), and examples include infection (ie, *Clostridioides difficile*), ischemia, or Crohn disease [1,8,11-14]. (See 'Differential diagnosis' below.)

ETIOPATHOGENESIS

The pathogenesis of acute pouchitis is unclear, but it is hypothesized to result from an abnormal immune response to altered luminal and/or mucosal bacteria in genetically susceptible hosts. Pouchitis is not an isolated disease entity. Instead, it likely represents a disease spectrum ranging from acute antibiotic-responsive pouchitis to chronic antibiotic-refractory pouchitis. These disease phenotypes may reflect the same disease process at different stages or various disease processes with different etiopathogenetic pathways.

Research studies on the pathogenesis of pouchitis have been limited because of the lack of an animal model with underlying inflammatory bowel disease. Thus, knowledge on the mechanism of disease in pouchitis has been largely derived from observations from clinical practice.

Genetic factors — Pouchitis occurs predominantly in patients with restorative proctocolectomy with underlying inflammatory bowel disease and less commonly in patients with familial adenomatous polyposis (FAP), suggesting the contribution of genetic factors to its pathogenesis. Immunogenetic studies have demonstrated that genetic polymorphisms such as those of interleukin (IL)-1 receptor antagonist, IL-1-beta, and NOD2/CARD15 or a combined carriership of TLR9-1237C and CD14-260T alleles were associated with an increased risk for acute or chronic pouchitis [15-20]. Similarly, mutations of NOD2/CARD15 and TNFSF15 have been associated with higher risk for severe pouchitis [21]. In addition, lower levels of expression of transcripts of genes related to xenobiotic efflux have been found in patients with pouchitis, suggesting a role of barrier dysfunction in inflammation of the pouch [22].

Intestinal microbiota — The intestinal microbiota has been linked to the development of pouchitis via the following mechanisms (see "Spatial organization of intestinal microbiota in health and disease"):

- Quantity of bacteria – The quantity of bacteria in the pouch and small bowel may contribute to symptoms and mucosal inflammation in patients with ileal pouches. Theoretically, all patients with ileal pouches have some degree of bacterial overgrowth in the small intestine, mainly due to the absence of a valve structure between the pouch body and afferent bowel limb. In addition, fecal stasis can result from structural or functional pouch outlet obstruction. However, diagnostic evaluation with carbohydrate

breath testing for small intestinal bacterial overgrowth may not be reliable in patients with ileal pouch-anal anastomosis, because the normal reference values are based on results for patients with an intact ileocecal valve. (See "Small intestinal bacterial overgrowth: Clinical manifestations and diagnosis".)

- Microbial diversity – Dysbiosis, which is often measured by bacterial diversity, has been reported in patients with pouchitis. Decreased bacterial diversity has been observed more often in patients with a history of ulcerative colitis (UC) rather than FAP [23-25]. Studies have demonstrated that Firmicutes families and Proteobacteria were increased, while *Ruminococcaceae* family was reduced in UC pouches as compared with FAP pouches [23]. In addition, a longitudinal study showed the reduction in genus *Ruminococcus* may precede the development of pouchitis [25].

  However, no individual family, genus, or species has been consistently linked to risk for pouchitis. Nonetheless, some data have suggested that bacterial species such as Lachnospiraceae, *Incertae Sedis XIV*, and *Clostridial* cluster IV have been implicated in the development of pouchitis [26-29]. Studies have also suggested that Bacteriodaceae species and Clostridiaceae species may be related to pouch inflammation, while Enterococcaceae species may play a role in maintaining immune homeostasis in the pouch mucosa [30]. Another study found increased proportions of the Fusobacteriaceae family and decreased *Faecalibacterium* in patients with pouchitis [31].

  The composition of mucosa-associated microbiota has been linked to pouchitis [32]. However, the microbiome and host transcriptome during pouchitis are affected by biopsy location and the presence of the inflammation [33] as well as the use of antibiotics [31,33] and fecal microbiota transplantation [33].

  The colon-like mucosa also creates a suitable environment for harboring colonic microbiota. For example, colonic metaplasia in the pouch predisposes to the growth of sulfate-reducing bacteria that produce hydrogen sulfide, which in high concentrations is toxic to colonocytes [27]. A gradual shift from an ileum-like to a colon-like bacterial community (including nonculturable bacteria such as *Clostridium coccoides* strains) occurs in the ileal pouch after colectomy [34]. Although the phylogenetic composition of the fecal microbiota community is stable in healthy individuals, the construction of an ileal pouch promotes fecal stasis with bacterial overload and mucosal adaptive changes from small bowel mucosa to colon-like mucosa (ie, colonic metaplasia) of the pouch body and inflammation (picture 1) [35].

- Biosynthetic capability – Alterations in intestinal microbiota may affect bile acid production related to the biosynthetic capability of microbes, and this may result in intestinal inflammation [23,36]. As an example, reduced levels of secondary bile acids (lithocholic acid and deoxycholic acid), were associated with fecal samples from pouches in patients with UC compared with FAP [23]. Changes in the bile acid profile in pouches related to UC may be related to the reduction in *Ruminococcaceae* family.

- Microbial pathogens – For some patients, pouchitis has been associated with infection with specific bacterial, viral, or parasitic pathogens, with *C. difficile* infection being most common and possibly related to repeated or chronic exposure to antibiotics [11,37,38]. Other pathogenic microbes that have been less commonly associated with active pouchitis include cytomegalovirus, *Clostridium perfringens, Campylobacter* spp, Group D Streptococci (*Enterococcus* spp), hemolytic strains of *E. coli*, and *Histoplasma capsulatum* [12,39-45].

Abnormal mucosal immunity — Pouchitis has been associated with an alteration in both innate and adaptive mucosal immunity [46-52].

Fecal stasis with microbial overload in the ileal pouch reservoir can cause adaptive morphologic changes in the pouch mucosa, resulting in colonic metaplasia [53]. Altered mucin glycoproteins may be more susceptible to enzymatic degradation by microbiota, leading to the breach of mucosal barrier function and subsequent pouchitis [54]. (See 'Intestinal microbiota' above.) Prior to the onset of histologic inflammation, pouches in patients with UC were shown to display a system-level gain of colon-associated gene profile and loss of ileum-associated gene profile [55].

Mucosal infiltration of acute (eg, neutrophils or eosinophils) and chronic (eg, mononuclear cells) inflammatory cells has been commonly seen in pouchitis (picture 1). However, the role of these acute and chronic inflammatory cells in the pathogenesis of pouchitis has been poorly defined.

Aberrant expression of Toll-like receptors (TLR) has also been described in patients with ileal pouches, while higher mucosal TLR expression has been associated with chronic pouchitis [52,56,57]. The increase in mucosal permeability to microbiota in the ileal pouch results in a mucosal immune response, as suggested by the production of antimicrobial peptides by intestinal Paneth cells and other gut epithelial cells [47,52,56-60]. The expression of Paneth-cell's human defensin-5 is increased in both inflamed and noninflamed pouches compared with normal terminal ileum [61-63]. In addition, aberrant expression of human beta defensin-1 has been observed in patients with pouchitis [64].

The microRNA expression and processing were increased in the inflamed intestinal mucosa of patients with pouchitis. The micro RNAs are regulators of gene expression at the posttranscriptional level by regulating mRNA stability and translation and play a role in cell proliferation, differentiation, and death [65]. Furthermore, the expression of microRNA 21 and 223 was higher, whereas that of microRNA 192 and 196a was lower in pouchitis compared with noninflamed pouches [66].

Abnormalities in both molecular and cellular components of the gut adaptive immune system have been associated with pouch inflammation. However, the abnormal adaptive mucosal immunity likely represents the activation of nonspecific inflammatory cascades or pathways [67]. Changes in adaptive immunity have included the increased proliferation of immature plasma cells in inflamed pouches in patients with UC [50,51,68] and production of proinflammatory mediators (eg, tumor necrosis factor, cell adhesion molecules, platelet-activating factor, lipoxygenase products of arachidonic acids, vascular endothelial growth factor, and proinflammatory neuropeptides) [67,69-78]. In addition, an imbalance in the production of proinflammatory (eg, IL-8) and immunoregulatory (eg, IL-10) cytokines has been reported [71]. Based on these observations, therapies that target proinflammatory mediators (eg, anti-TNF agents, anti-interleukin and anti-integrin agents) have been studied for treating chronic pouchitis [79-86]. (See "Management of acute and chronic pouchitis", section on 'Immune-mediated chronic pouchitis'.)

RISK FACTORS

Ulcerative colitis (UC) — Clinical characteristics of UC associated with pouchitis include:

- **Disease extent** – Extensive colitis has been associated with an increased risk of pouchitis [87-90]. In a study including 201 patients with ileal pouch-anal anastomosis (IPAA) with a mean follow-up of nine years, patients with extensive UC had a threefold increased risk of chronic pouchitis compared with patients without extensive disease (odds ratio [OR] 3.3, 95% CI 1.2-8.9) [88]. (See "Medical management of low-risk adult patients with mild to moderate ulcerative colitis", section on 'Defining disease extent'.)

  However, backwash ileitis (ie, ileal inflammation in patients with UC) has been inconsistently associated with risk of pouchitis [91,92].

- **Extraintestinal manifestations** – Extraintestinal manifestations including primary sclerosing cholangitis are risk factors for pouchitis [4,87,88,93-95]. In a study that included 1097 patients with UC, of whom 54 had primary sclerosing cholangitis (PSC), the

cumulative risk of pouchitis after IPAA in patients with both UC and PSC was higher at 1, 2, 5, and 10 years compared with UC alone (22, 43, 61, and 79 percent versus 16, 23, 36, 46 percent, respectively) [4]. In addition, patients with a PSC often have diffuse pouchitis and enteritis of a long segment of the afferent limb [96,97].

- **Age at diagnosis or surgery** – Younger age at the time of diagnosis of UC or IPAA surgery has been associated with an increased risk of pouchitis [98-100]. Possible mechanisms contributing to risk in pediatric patients include pull-through surgical technique with a long rectal cuff, the smaller size of the pouch body, and accumulation of peripouch mesenteric tissue that may compromise pouch function. (See "Management of the hospitalized child or adolescent with acute severe ulcerative colitis", section on 'Surgery'.)

Coexisting autoimmune disorders — Autoimmune-mediated disorders (eg, high serum IgG4, autoantibodies to the host's tissue such as perinuclear antineutrophil cytoplasmic antibodies [p-ANCA]) have been associated with an increase in the risk of pouchitis in some studies [88,101-108]. In a meta-analysis including 335 patients with UC who had undergone IPAA and had acute or chronic pouchitis, positive serology for ANCA was associated with an increased risk for chronic pouchitis compared with negative ANCA (OR 1.8, 95% CI 1.2-2.6) [109]. However, ANCA titers were measured following colectomy in most of the studies included in the meta-analysis, and the analysis did not adjust for confounding variables that can increase the risk of pouchitis.

IgG4-related disease has been associated with risk for antibiotic-refractory pouchitis, and such patients are often managed with therapies that modify the immune response. (See "Management of acute and chronic pouchitis", section on 'IgG4-associated chronic pouchitis' and "Treatment and prognosis of IgG4-related disease".)

Metabolic factors — Obesity (body mass index ≥30 kg/m^2) has been identified as a risk factor for inflammatory disorders of the pouch in patients with restorative proctocolectomy [110,111]. Postoperative weight gain was associated with a high risk for pouch failure from chronic pouchitis, Crohn disease of the pouch, or mechanical complications [112]. Patients with an ileal pouch for UC were found to have more peripouch fat as measured with cross-sectional imaging compared with patients with familial adenomatous polyposis [113,114].

Lifestyle factors — Lifestyle factors that have been linked to the risk for pouchitis include:

- **Smoking status** – Smoking has been associated with an increased risk of acute antibiotic-responsive pouchitis, possibly because of the effects of tobacco on the intestinal microbiome [115,116]. However, the effect of smoking on chronic pouchitis is uncertain, and cigarette smoking may affect the clinical course of related conditions such as UC. (See

"Definitions, epidemiology, and risk factors for inflammatory bowel disease", section on 'Clinical risk factors'.)

- **Dietary factors** – Dietary intake for patients with ileal pouches may vary from the general population, and diet may influence the composition of intestinal microbiota. In a cohort study including 172 patients with ileal pouches, lower fruit consumption was correlated with higher rates of pouchitis compared with higher fruit intake (31 versus 4 percent) [117]. In a cross-sectional study including 80 patients with ileal pouches, patients with pouches consumed more bakery products, oils, fats, nuts, and seeds compared with healthy controls [118]. (See 'Intestinal microbiota' above.)

Medication use — Observational studies have linked the following medications with the risk for pouchitis:

- **Nonsteroidal antiinflammatory drug (NSAID) use** – Postoperative NSAID use has been associated with an increased risk of chronic pouchitis [87]. Discontinuing NSAIDs in patients with ileal pouch disorders has been associated with improved symptoms [13].

- **Preoperative UC treatment** – Some medical therapies for UC have been linked to pouchitis; however, this observation may reflect severity of underlying disease [10]:

  - Glucocorticoids – High cumulative dose of glucocorticoids (>10,000 mg) prior to proctocolectomy has been associated with an increased risk for pouchitis [119].

  - Anti-tumor necrosis factor (TNF) agents – Preoperative use of an anti-TNF agent has been linked to an increased risk for pouchitis. In a study including 417 patients who underwent IPAA, use of anti-TNF therapy before surgery was associated with an increased risk for pouchitis (48 versus 37 percent, OR 1.52; 95% CI 1.00-2.30) [120].

Other factors

- **Pouch configuration** – Some pouch configurations have been associated with an increased risk for pouchitis, while the J-shaped pouch operation is most commonly performed due to its greater ease of construction [121]. In a study of 215 patients with J-shaped pouches and 45 patients with S-shaped pouches, S-pouches were associated with a lower risk of chronic pouchitis compared with J-pouches (OR 0.07, 95% CI <0.001-0.54) [122]. This observation likely resulted from lower mesenteric tension (and thus, less risk of ischemia) from S-pouch construction. In a systemic review of seven studies including 548 patients with an ileal pouch, the risk of pouchitis was not significantly different for patients with J-shaped pouches compared with W-shaped pouches; however, W-shaped pouch

construction is no longer performed at most centers [121]. (See "Surgical management of ulcerative colitis", section on 'Surgical options'.)

- **Other surgical factors** – A history of multiple abdominal and/or pelvic surgeries has been associated with pouchitis, possibly due to poor perfusion or ischemia during or after pouch construction [123]. In addition, a strictured anastomosis is associated with a higher risk for pouchitis, presumably due to backflow of stool content and ischemia [124].

- **Hematologic disorders** – Perioperative thrombocytosis and portal vein thrombosis have been associated with an increased risk of pouchitis, presumably due to ischemia [123,125].

- **Fecal stasis** – Fecal stasis resulting from structural or functional pouch outlet obstruction has been associated with pouchitis [1].

CLINICAL FEATURES

Clinical features of pouchitis include:

- **Patient presentation** – Pouchitis has a variety of clinical phenotypes, and patients may have acute or chronic disease with or without a relapsing pattern. The spectrum of presentation also includes a range of symptom severity, from an increase in stool frequency above baseline (normally four to seven bowel movements daily) with urgency to more debilitating symptoms including pelvic pain and incontinence. Other common symptoms of pouchitis include abdominal cramping and pelvic pressure. In addition, patients with pouchitis can present at any age.

- **Physical examination** – For some patients, physical examination may be normal; however, the examination may demonstrate tenderness of lower abdomen, hyperactive bowel sounds, and perianal dermatitis.

- **Laboratory findings** – Routine laboratory tests may be normal or they may reveal anemia, an elevated C-reactive protein, electrolyte abnormalities, iron deficiency, and vitamin D deficiency [126,127].

  Stool inflammatory markers (fecal calprotectin or lactoferrin) may be elevated due to intestinal inflammation [128,129].

  Patients with coexisting primary sclerosing cholangitis or IgG4-associated cholangiopathy may have an elevation of alkaline phosphatase and/or bilirubin. (See "Primary sclerosing cholangitis in adults: Clinical manifestations and diagnosis", section on 'Association with

inflammatory bowel disease' and "Pathogenesis and clinical manifestations of IgG4-related disease", section on 'IgG4-related sclerosing cholangitis'.)

- **Imaging findings** – Radiographic findings suggestive of acute pouchitis on cross-sectional imaging include thickening of the pouch wall, mucosal hyperenhancement, accumulation of peripouch fat, and pelvic lymphadenopathy (image 1). However, imaging is not routinely obtained for patients with suspected acute idiopathic pouchitis.

EVALUATION

Goals of the diagnostic evaluation for patients with suspected active idiopathic pouchitis are to exclude other causes of symptoms, establish the diagnosis of pouchitis, and determine the severity of the disease. (See 'Scoring systems for assessing disease activity' below.)

History — The history serves to identify symptoms that are suggestive of other conditions:

- Rectal bleeding – Bleeding occurs rarely in patients with pouchitis and is more suggestive of other disorders such as inflammation of the rectal cuff (cuffitis) and pouch prolapse.
- Systemic symptoms – Systemic symptoms including fever, chills, sweats, and weight loss are suggestive of an infectious cause of pouchitis (eg, *Clostridioides difficile*), Crohn disease (CD) of the pouch, or surgical complications (eg, pouch anastomotic leak).
- Straining or ineffective defecation – Dyschezia (ie, ineffective defecation manifested as straining in the absence of constipation) is suggestive of an obstructive disorder such as an anastomotic stricture, distal pouch prolapse, or dyssynergic defecation. (See "Etiology and evaluation of chronic constipation in adults", section on 'Outlet delay'.)

  While patients with acute pouchitis may have a sense of incomplete evacuation, they typically do not report straining.

Approach to testing — The typical workup for most patients in whom acute pouchitis is suspected includes:

- Laboratory studies including blood tests and stool studies
- Endoscopic evaluation of the pouch (pouchoscopy) and mucosal biopsy

Laboratory studies — For patients with suspected acute pouchitis, we obtain the following laboratory studies [103,105,108]:

- Blood tests – Blood tests include complete blood count, basic metabolic panel, aspartate aminotransferase, alanine aminotransferase, total bilirubin, alkaline phosphatase, and C-reactive protein.

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

  - For patients with increased stool frequency above baseline (≥8 stools daily), a stool specimen is sent to exclude *Clostridioides difficile* infection [130-132]. Stool testing for *Clostridioides difficile* infection is discussed separately. (See "*Clostridioides difficile* infection in adults: Clinical manifestations and diagnosis", section on 'Diagnosis'.)

  - For patients with ≥8 stools daily and systemic symptoms such as fever, chills, and sweats, stool cultures for other intestinal pathogens (eg, *Salmonella, Shigella, Campylobacter, Yersinia*) and specific testing for *E. coli* O157:H7 are also performed [85]. (See "Approach to the adult with acute diarrhea in resource-abundant settings", section on 'Evaluation'.)

    For patients without systemic symptoms, we do not routinely obtain additional stool studies (eg, testing for other intestinal pathogens) because the yield of such testing is low [133].

- Stool inflammatory markers – Although tests for fecal calprotectin or lactoferrin are not used routinely to diagnose pouchitis and do not replace endoscopic evaluation, they may help to differentiate patients with intestinal inflammation from patients with irritable pouch syndrome [128]. While calprotectin is used more commonly by some clinicians, lactoferrin is an acceptable alternative [128,129]. (See "Approach to the adult with chronic diarrhea in resource-abundant settings", section on 'General laboratory tests'.)

Endoscopy with biopsy — Endoscopy of the ileal pouch (including mucosal biopsies) and prepouch ileum is performed for the evaluation of suspected acute pouchitis [1,32,134], and examination includes:

- Endoscopic visualization of the pouch – Endoscopic features of active pouchitis include diffuse erythema, friability, granularity, exudates, erosions, and/or ulcerations [134,135]. For most patients with active pouchitis, mucosal inflammation with or without ulceration is typically distributed throughout the pouch body (picture 2). Some patients may have a short segment (<10 cm) of concurrent inflammation at the distal afferent limb.

  The severity, extent, and distribution of mucosal inflammation of the pouch is reported, in addition to configuration, size, and distensibility of the pouch body.

Inflammatory polyps, poor distensibility of the pouch body, or the loss of "owls' eye" configuration of a J-pouch may be seen in patients with a chronically inflamed ileal pouch (picture 3) [136,137].

- Biopsies of the pouch – During pouchoscopy, mucosal biopsies of the pouch are obtained; however, biopsies from the suture-line are avoided because histologic evaluation may yield granulomas that may mimic CD-associated granulomas [138].

  On histology, pouchitis is characterized by acute inflammation with neutrophil infiltration, crypt abscess, and mucosal ulceration often superimposed on a background of chronic changes, including villous atrophy, crypt distortion, or hyperplasia, pyloric gland metaplasia, and chronic inflammatory cell infiltration (picture 1) [139,140].

  Histology showing crypt apoptosis is suggestive of chronic, autoimmune-mediated pouchitis [141,142].

- Endoscopic assessment of the prepouch ileum – The prepouch ileal mucosa (ie, distal afferent limb of ileum) is assessed endoscopically for inflammatory changes (eg, erythema, ulceration). Some patients with pouchitis (eg, those with primary sclerosing cholangitis) can have coexisting disease involving a long segment (≥10 cm) of the distal afferent limb (ie, prepouch ileitis/enteritis) [143]. (See "Endoscopic diagnosis of inflammatory bowel disease in adults", section on 'Ileocolonoscopy'.)

- Perianal inspection and digital rectal examination – Prior to pouchoscopy, perianal inspection and digital rectal examination are performed to evaluate for other conditions such as rectal stricture, anal fissure, or perianal fistula. (See "Perianal Crohn disease", section on 'Initial evaluation of symptoms'.)

Other diagnostic testing — We do not routinely obtain imaging (eg, cross-sectional imaging, pouchogram with water-soluble contrast) for patients with suspected acute idiopathic pouchitis because the diagnosis of acute idiopathic pouchitis is largely based on the patient's symptoms and endoscopic findings. For patients with symptoms that are suggestive of other pouch disorders such as anastomotic stricture or pouch prolapse, diagnostic imaging is typically obtained. (See 'Differential diagnosis' below.)

Scoring systems for assessing disease activity — Several scoring instruments have been used to grade the severity of inflammation in patients with pouchitis in research studies, while these indices are used less often in routine clinical practice [139,144-147]. A commonly used index is the Pouchitis Disease Activity Index (PDAI), which consists of symptom, endoscopic, and histologic subscores (table 1) [139,148]. Active pouchitis has been defined as PDAI score of ≥7

points [149]. However, the PDAI is not specific for idiopathic pouchitis because other inflammatory disorders of the pouch (eg, CD of the pouch) can also result in an elevated PDAI score. (See 'Differential diagnosis' below.)

A modified version of the PDAI (ie, modified Pouchitis Disease Activity Index [mPDAI]) is an alternative instrument that does not include a histology subscore. Active pouchitis has been defined as mPDAI score of ≥5 [144]. The mPDAI has also been used in clinical trials [81,145,150].

DIAGNOSIS

The diagnosis of acute pouchitis should be suspected in patients with ileal pouch-anal anastomosis and compatible clinical features such as increased stool frequency above baseline, urgency, and abdominal cramping.

The diagnosis of acute pouchitis is established with endoscopic and histologic findings that demonstrate active inflammation of the pouch (eg, mucosal erythema, ulceration, friability) in a patient with compatible clinical presentation (eg, increased stool frequency, urgency) [1]. Laboratory testing is complementary in assessing for other conditions (eg, bacterial infection), but does not establish the diagnosis of pouchitis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pouchitis is broad and includes (picture 2) [151]:

- **Postoperative complications** – Postoperative complications typically occur within the first year after ileal pouch construction and may be due to fistula development, pelvic sepsis, and/or mechanical issues affecting pouch emptying. Specific conditions include [152] (see "Restorative proctocolectomy with ileal pouch-anal anastomosis: Laparoscopic approach"):
  - Postoperative pelvic infection – For patients who develop symptoms of fever, drainage and/or pelvic pain shortly after pouch construction and ileostomy closure, postoperative complications such as pouch anastomotic leak or pelvic abscess should be suspected. Chronic leaks typically present with pouch fistula or sinus. Cross-sectional imaging is typically performed when abdominal or pelvic abscess or fistula is suspected. In addition, patients with a history of postoperative pelvic infection may develop decreased compliance with the ileal pouch. Patients may present with diarrhea, urgency, and/or incontinence, and have evidence of decreased compliance on imaging (barium pouchogram and/or pelvic magnetic resonance imaging) [153].

- Anastomotic stricture – Postoperative mechanical issues such as anastomotic stricture can be diagnosed by digital rectal examination, while pouch strictures are identified endoscopically or with an imaging study (ie, water contrast or barium pouchogram). An anastomotic stricture can result in excessive fecal stasis and bacterial overload, leading to the development of stasis-associated pouchitis, and a procedural intervention to relieve the obstruction is often needed [154].

- **Pouch prolapse** – Pouch prolapse can be detected based on endoscopic visualization (pouchoscopy) that demonstrates mucosal edema, erythema, and a distorted pouch inlet area (picture 3). Pouch prolapse may be limited to the mucosa or may be a full-thickness prolapse, and it is commonly located at the anterior wall of distal pouch. Pouch prolapse is one form of floppy pouch complex [153,155].

- **Ischemic pouchitis** – Ischemic pouchitis is characterized endoscopically by asymmetric inflammation and ulceration of the pouch. Mucosal disease affecting one half of the pouch with demarcation along the suture line suggests ischemia or inflammation may involve the distal pouch or the afferent limb only. On histology, ischemic pouchitis is characterized by extracellular hemosiderin or hematoidin deposits [123,137]. Risk factors include male sex and weight gain, possibly leading to mesenteric tension. Nonsurgical therapies for ischemic colitis have included hyperbaric oxygen therapy [156] and vedolizumab (based on author's experience).

- **Cuffitis** – Classic cuffitis is a recurrence of ulcerative colitis (UC) in the residual cuff of rectal mucosa following proctocolectomy with IPAA. Patients with cuffitis typically present with hematochezia, while rectal bleeding occurs rarely in patients with active pouchitis only [157]. On pouchoscopy, cuffitis appears as circumferential inflammation of the rectal cuff with histologic findings consistent with UC. Non-classic cuffitis may result from other etiologies, such as Crohn disease, surgical ischemia, and prolapse. (See "Clinical manifestations, diagnosis, and prognosis of ulcerative colitis in adults", section on 'Endoscopy and biopsy'.)

- **Crohn disease (CD) of the pouch** – CD of the pouch can occur in patients with a preoperative diagnosis of UC (ie, new-onset CD of the pouch) [158]. CD of the pouch may also develop in patients with an established preoperative diagnosis of Crohn colitis or with an incidental diagnosis of Crohn colitis based on the colectomy specimen [159]. CD of the pouch may have an inflammatory, fibrostenotic, or fistulizing phenotype, similar to luminal CD. Patients with CD of the pouch usually have segmental inflammation of the pouch body and/or afferent limb. In addition, patients with CD may have strictures at the pouch inlet or afferent limb, complex perianal fistulas, or late-onset (>6 to 12 months after ileostomy

closure) pouch-vaginal fistulas with an internal fistula opening at the anal canal area. Clinical features suggestive of CD of the pouch include non-caseating, non-crypt-rupture-associated granulomas on intestinal biopsy of the afferent limb, pouch body, or cuff; segmental or skip lesions or strictures in the pouch or small bowel; late development of fistulas or abscess (ie, 6 to 12 months after stoma closure); and pre-pouch ileitis [1]. (See "Perianal Crohn disease", section on 'Perianal fistula'.)

- **Prepouch ileitis** – Some symptoms of prepouch ileitis (eg, increased stool frequency) may overlap with active pouchitis, while other symptoms of ileitis include obstructive symptoms (eg, straining) and bleeding [160]. In addition, prepouch ileitis may coexist with pouchitis, and may be related to backwash from diffuse pouchitis with a patulous inlet or possibly related to ischemia [161,162]. Prepouch ileitis may be related to a specific etiology (eg, CD, NSAID-related injury, ischemia). Medical therapy with biologic agents has demonstrated limited efficacy, and some patients have required surgical management for pouch failure [161].

- **Diversion pouchitis** – Patients with diversion pouchitis present with anal discharge of blood and/or mucus and fecal urgency. Diversion pouchitis only develops in patients with an ileal pouch-anal anastomosis with a diverting ileostomy.

- **Irritable pouch syndrome** – Irritable pouch syndrome (IPS) is a functional disorder associated with visceral hypersensitivity that is characterized by diarrhea, abdominal cramping, and urgency without endoscopic or histologic inflammation of the pouch [163]. Histologic evaluation may show enterochromaffin cell hyperplasia on pouch biopsy; however, this is not required to diagnose IPS [164]. The pathophysiology and diagnosis of functional bowel disorders are discussed separately. (See "Pathophysiology of irritable bowel syndrome" and "Clinical manifestations and diagnosis of irritable bowel syndrome in adults".)

- **Infectious pouchitis** – Symptoms of an infectious process include increased stool frequency, pelvic pain, and/or fever, and *Clostridioides difficile* is a common cause of acute infectious pouchitis, while cytomegalovirus (CMV) is rarely associated with pouchitis [10,12,37]. Stool testing for *Clostridioides difficile* can help exclude this pathogen, and the diagnosis and management of *Clostridioides difficile* infection are discussed separately. (See "*Clostridioides difficile* infection in adults: Clinical manifestations and diagnosis", section on 'Diagnosis' and "*Clostridioides difficile* infection in adults: Treatment and prevention".)

The diagnosis of CMV infection is based on the presence of CMV inclusion bodies or positive immune histochemistry, and this is discussed in more detail separately. (See "Epidemiology, clinical manifestations, and treatment of cytomegalovirus infection in immunocompetent adults", section on 'Gastrointestinal manifestations' and "Approach to the diagnosis of cytomegalovirus infection".)

- ***De novo* celiac disease** – Celiac disease can develop *de novo* after restorative proctocolectomy and ileal pouch-anal anastomosis [165]. Serology for celiac disease is obtained during the initial evaluation of suspected pouchitis, even if preoperative testing was normal. If serology is positive, duodenal biopsy is performed to confirm the diagnosis [166]. The diagnosis and management of celiac disease are discussed separately. (See "Diagnosis of celiac disease in adults" and "Management of celiac disease in adults".)

PROGNOSIS

Most patients with acute pouchitis initially have symptomatic resolution with a course of antibiotics; however, approximately 50 to 90 percent of patients will have at least one recurrence and approximately 30 percent of patients will progress to chronic pouchitis [24,93,167,168]. As an example, in a study of 386 patients with proctocolectomy and ileal pouch-anal anastomosis, 205 patients (53 percent) developed acute pouchitis, of whom 60 patients (30 percent) progressed to chronic pouchitis during a median follow-up of four years [167].

The onset of acute pouchitis immediately after pouch construction and ileostomy closure has been associated with an increased risk for developing chronic pouchitis [169]. It has been estimated that up to 20 percent of patients with acute pouchitis develop chronic antibiotic refractory pouchitis (CARP) [168,170-172]. CARP may progress to pouch failure, and risk factors and surgical management of pouch failure (eg, permanent diversion with ileostomy) are discussed separately. (See "Restorative proctocolectomy with ileal pouch-anal anastomosis: Laparoscopic approach", section on 'Risk factors for pouch failure'.)

Long-term functional results for patients with ileal pouch are discussed separately. (See "Restorative proctocolectomy with ileal pouch-anal anastomosis: Laparoscopic approach", section on 'Functional results'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Pouchitis".)

SUMMARY AND RECOMMENDATIONS

- **Background** – Restorative proctocolectomy with ileal pouch-anal anastomosis (IPAA) is often performed for patients with ulcerative colitis (UC) or familial adenomatous polyposis who require colectomy. However, patients with IPAA are at risk for pouchitis, an inflammatory disorder that typically presents with increased stool frequency and urgency and is a common complication of IPAA. (See 'Introduction' above.)

  Many patients with ileal pouches have some degree of histologic inflammation of the ileal pouch reservoir, and pouchitis likely represents a disease spectrum with a range of presentations that may evolve over time. In addition, disease characteristics (eg, response to antibiotics, frequency of relapses) have been used to classify pouchitis and help guide treatment. As an example, acute antibiotic-responsive pouchitis may evolve into chronic antibiotic-refractory pouchitis. (See 'Spectrum of disease' above.)

- **Risk factors** – Risk factors for acute or chronic pouchitis have included characteristics of UC (disease extent, extraintestinal manifestations), coexisting autoimmune disorders (eg, primary sclerosing cholangitis), obesity, and medication use (eg, NSAIDs). (See 'Risk factors' above.)

- **Clinical features** – Symptoms of active pouchitis range in severity from an increase in stool frequency above baseline (normally four to seven bowel movements daily) with urgency to more debilitating symptoms including pelvic pain and incontinence. Other common symptoms include abdominal cramping, urgency, and pelvic pressure. (See 'Clinical features' above.)

- **Evaluation** – Goals of the diagnostic evaluation for patients with suspected acute idiopathic pouchitis are to exclude other causes of symptoms, establish the diagnosis of pouchitis, and determine the severity of the disease. The evaluation typically includes pouchoscopy with mucosal biopsy and laboratory studies (blood and stool tests [eg, testing for *Clostridioides difficile* toxin]). (See 'Evaluation' above.)

- **Diagnosis** – The diagnosis of acute pouchitis is suspected in patients with IPAA and compatible clinical features such as increased stool frequency above baseline and urgency. (See 'Diagnosis' above.)

  The diagnosis of acute pouchitis is established with endoscopic and histologic findings that demonstrate active inflammation of the pouch (eg, mucosal erythema, ulceration) in a patient with compatible clinical presentation (eg, increased stool frequency, urgency).

Laboratory testing is complementary in assessing for other conditions (eg, bacterial infection), but does not establish the diagnosis of pouchitis.

- **Prognosis** – Most patients with acute pouchitis initially have symptomatic resolution with a course of antibiotics; however, approximately 50 to 90 percent of patients will have at least one recurrence and approximately 30 percent of patients will progress to chronic pouchitis. (See 'Prognosis' above.)

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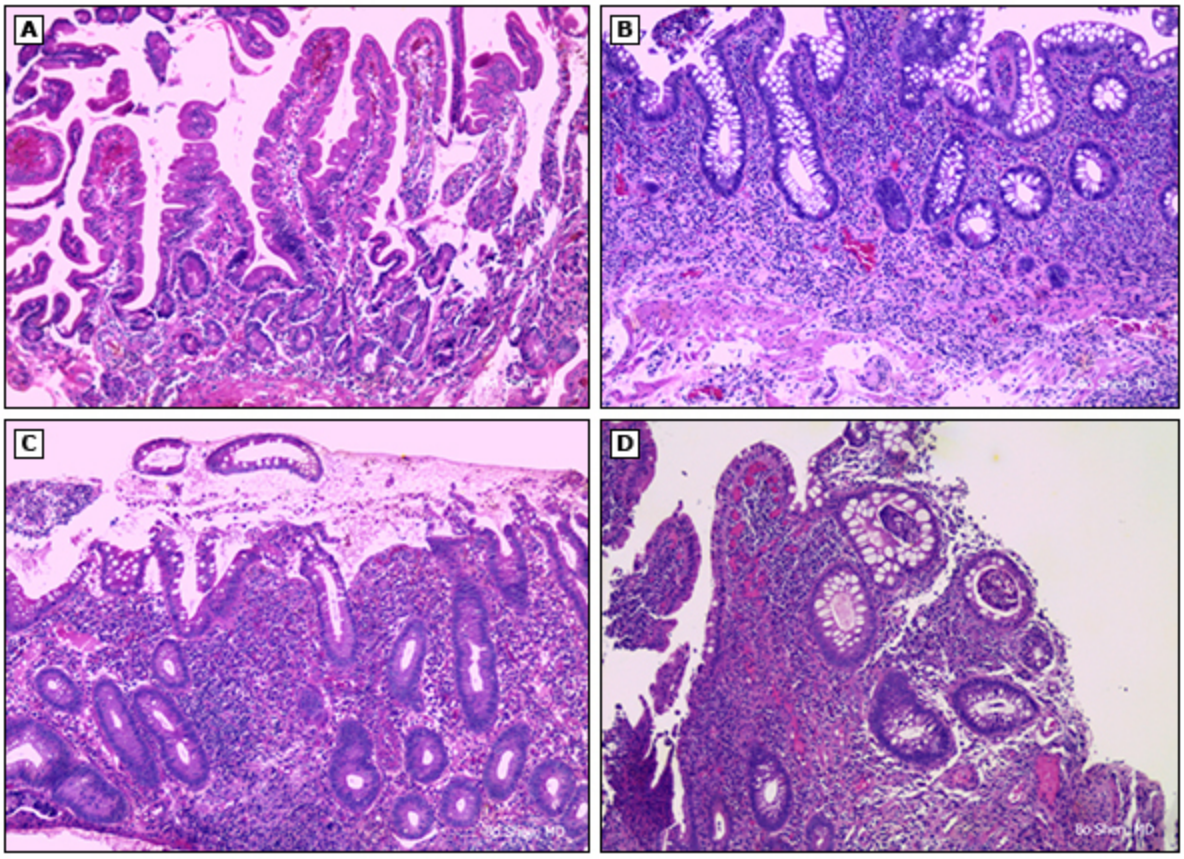
Topic 4073 Version 35.0

GRAPHICS

Histology of pouch mucosa



(A) Normal pouch mucosa with tall villi and normal villus/crypt ratio. No inflammatory cell infiltration in the epithelium and lamina propria.

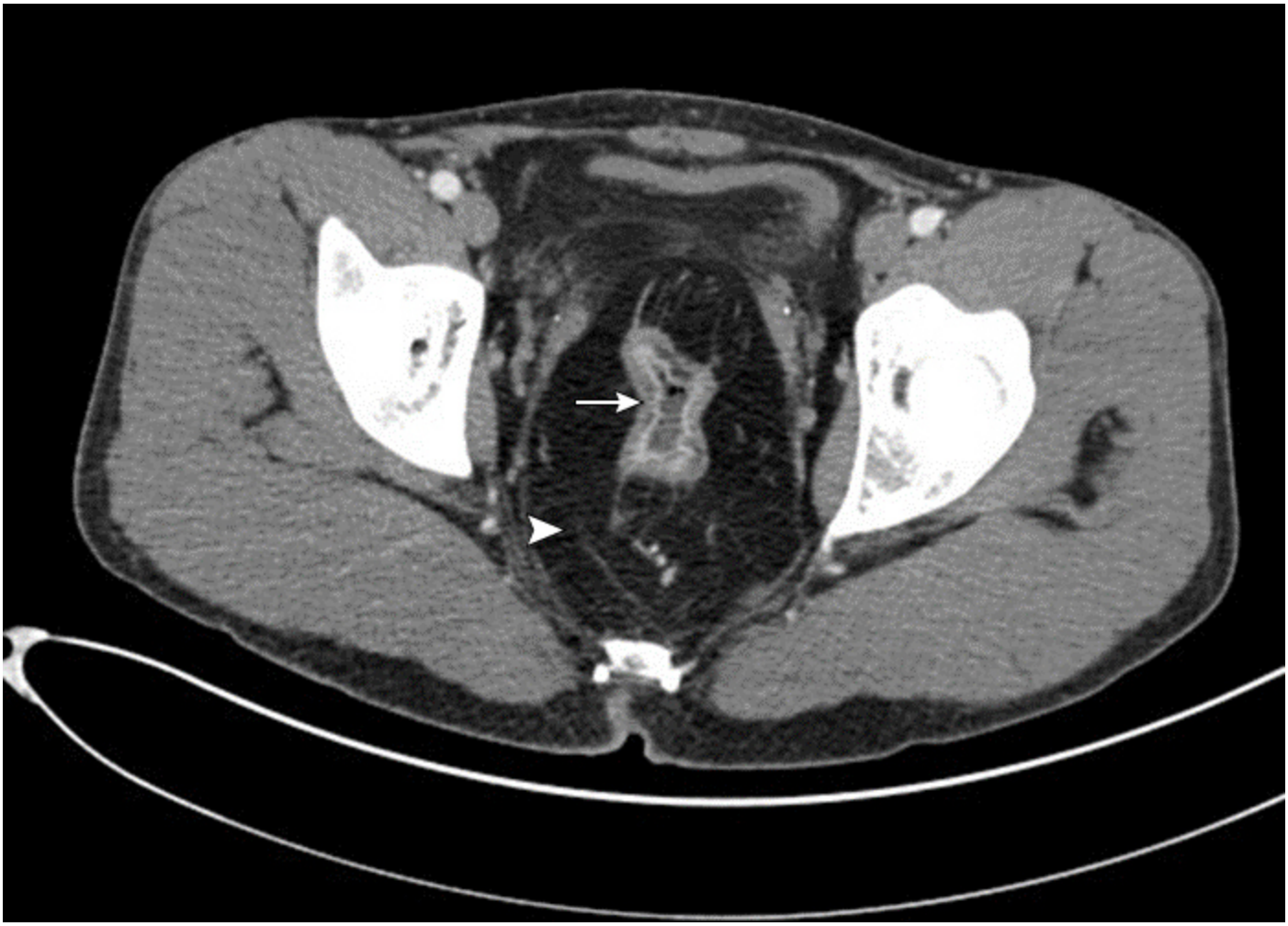
(B) Mild pouchitis with mainly chronic changes including crypt distortion, mononuclear cell infiltrations in the lamina propria and base of the crypts.

(C) Acute pouchitis with cryptitis and ulcers.

(D) Acute pouchitis with crypt abscess.

Graphic 93165 Version 2.0

Computed tomography demonstrating acute pouchitis

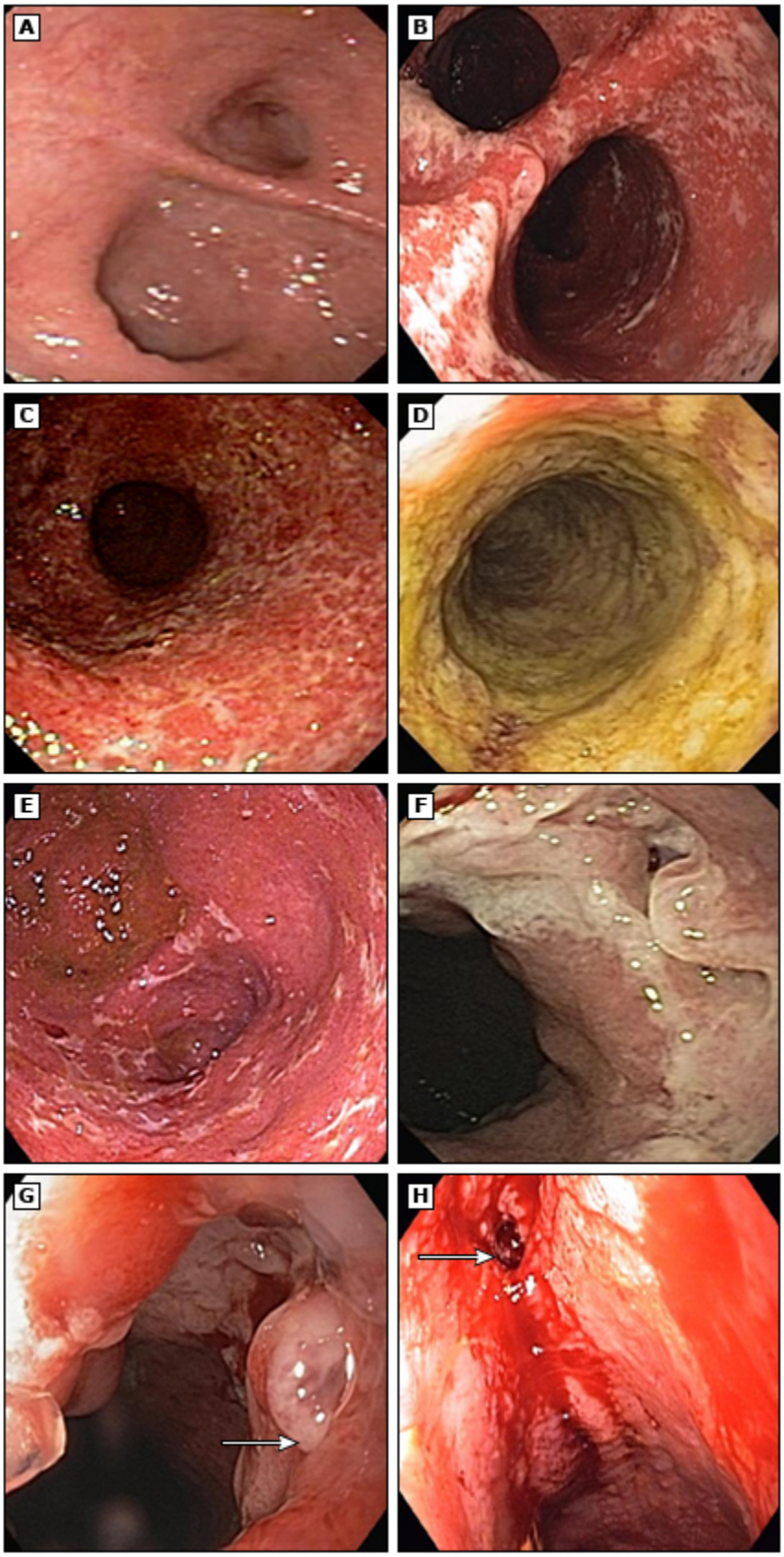


This computed tomography of the pelvis was performed in a patient with acute pouchitis. The pouch wall appears thickened with mucosal hyperenhancement (arrow). There is a large accumulation of peripouch fat (arrowhead).

Courtesy of Bo Shen, MD.

Graphic 129634 Version 1.0

Endoscopic spectrum of pouchitis and pouch disorders



(A) Normal pouch with owls' anatomy of the pouch inlet and the tip of "J".

(B) Diversion pouchitis.

(C) Classic pouchitis with diffuse inflammation.

(D) *Clostridium difficile* pouchitis with pseudomembrane.

(E) Ischemic pouchitis with asymmetric distribution of inflammation in the pouch body.

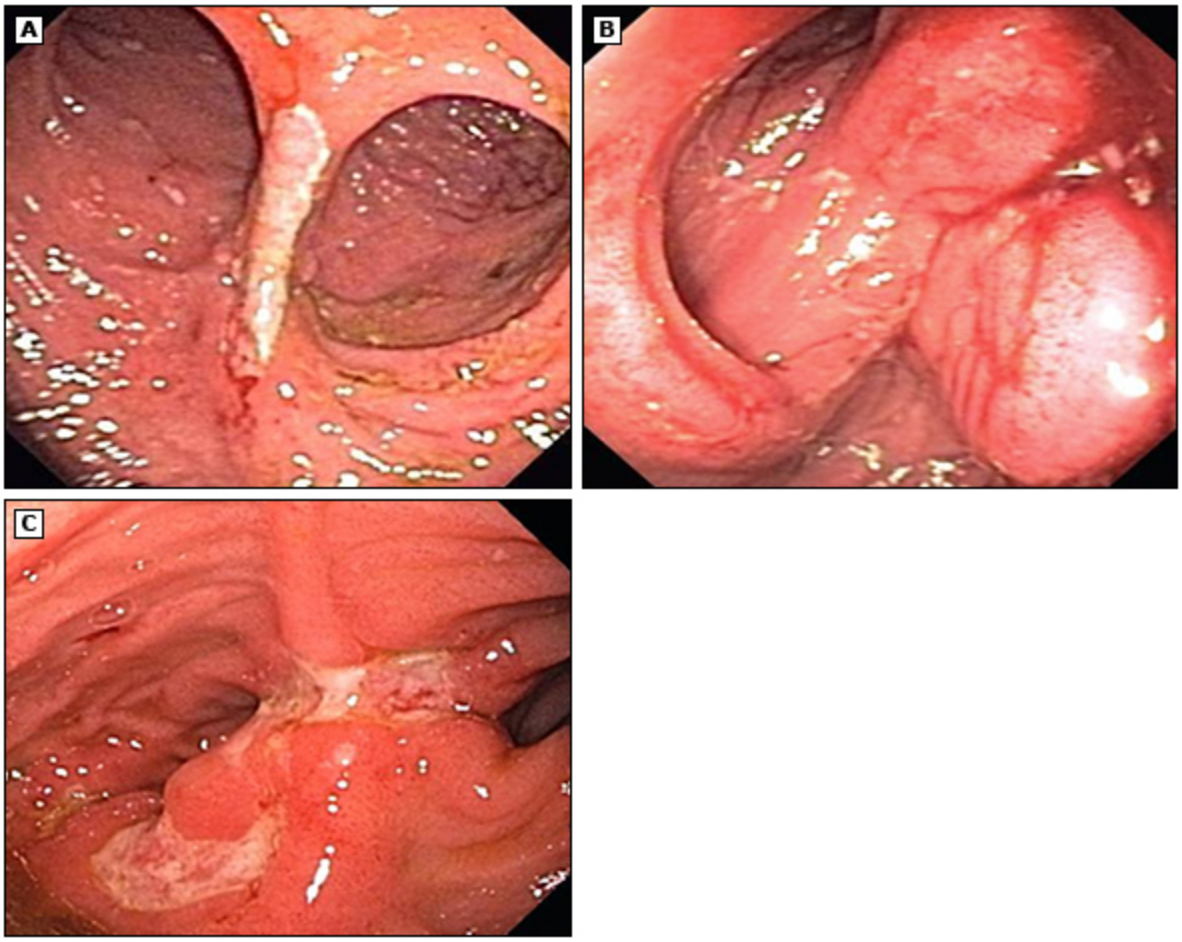
(F) Cuffitis.

(G) Crohn disease with pouch vaginal fistula opening (arrow).

(H) Anastomotic sinus due to surgical leak (arrow).

Graphic 93161 Version 1.0

Endoscopy of the "J" pouch



(A) Endoscopic view of a normal pouch inlet area (tip of "J" and inlet) with a central ridge has an owl's eye appearance.

(B) Distorted pouch inlet area in a patient with pouch prolapse.

(C) Distorted pouch inlet area in a patient with Crohn disease.

Graphic 93166 Version 1.0

Pouchitis Disease Activity Index (PDAI)

	Score
Clinical criteria	
Stool frequency	
Usual post-op stool frequency	0
One to two stools/day > post-op usual	1
Three or more stools/day > post-op usual	2

Rectal bleeding	
None or rare	0
Present daily	1
Fecal urgency/abdominal cramps	
None	0
Occasional	1
Usual	2
Fever (temperature >100°F)	
Absent	0
Present	1
Endoscopic criteria	
Edema	1
Granularity	1
Friability	1
Loss of vascular pattern	1
Mucus exudate	1
Ulceration	1
Acute histologic criteria	
Polymorph infiltration	
Mild	1
Moderate + crypt abscess	2
Severe + crypt abscess	3
Ulceration per low-power field (average)	
<25 percent	1
≥25 to ≤50 percent	2
>50 percent	3

Pouchitis is defined as a total PDAI score ≥7 points.

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Graphic 94138 Version 6.0

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