Communication from the ASGE IBD Endoscopy Consensus Panel

GUIDELINE

ASGE American Society for Gastrointestinal Endoscopy

# Endoscopic diagnosis and management of adult inflammatory bowel disease: a consensus document from the American Society for Gastrointestinal Endoscopy IBD Endoscopy Consensus Panel

**Prepared by: THE ASGE IBD ENDOSCOPY CONSENSUS PANEL**

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Endoscopy plays a key role in diagnosis, monitoring of disease activity, assessment of treatment response, dysplasia surveillance, postoperative evaluation, and interventional therapy for patients with inflammatory bowel disease (IBD). Clinical practice patterns in the endoscopic management of IBD vary. A panel of experts consisting of IBD specialists, endoscopists, and GI pathologists participated in virtual conferences and developed this modified Delphi-based consensus document to address endoscopic aspects of IBD management. (Gastrointest Endosc 2024;■:1-20.)

The incidence of inflammatory bowel diseases (IBD) is increasing worldwide.[1] The diagnosis and management of Crohn's disease (CD) and ulcerative colitis (UC) remain challenging, despite advances in medical, endoscopic, and surgical therapy. Accurate diagnosis, close disease monitoring and surveillance, and timely intervention are needed to improve patients' symptoms and quality of life. These can reduce the risk of tissue damage; development of adverse events, disease-associated hospitalization, and surgery; and overall healthcare burden.

Because of chronicity, extent, multifocal location, depth of the disease process, and altered bowel structure or anatomy, endoscopic assessment of IBD requires an understanding of the underlying disease and endoscopic techniques, combined with careful planning, preparation, and execution. Advances in endoscopic techniques, equipment, devices, and imaging can provide a better understanding, characterization, assessment, and management of IBD. However, the availability of high-quality, endoscopy-oriented clinical trials in IBD is lacking.

The American Society for Gastrointestinal Endoscopy (ASGE) organized a panel of experts in IBD, endoscopy, and GI pathology. A modified Delphi process was used to develop this clinically oriented, practical consensus document on the appropriate application and execution of diagnostic and therapeutic endoscopy in the management of IBD.

## METHODS

### Search strategy

The steering committee first reviewed the medical literature for each statement and generated key words for a literature search. A systematic search of MEDLINE, Google Scholar, EMBASE, and Cochrane Central Register of Controlled Trials from January 1, 2000 to July 31, 2024 was performed. Key search terms were "inflammatory bowel disease," "Crohn's disease," "ulcerative colitis," "pouchitis," "inflammatory," "stricture," "fistula," "dysplasia," "neoplasia," "stricture," "diversion," "postoperative," "ileostomy," "strictureplasty," "diagnosis," "therapy," "disease activity," "endoscopy," "colonoscopy," "ileostomy," "capsule endoscopy," and "enteroscopy."

### Consensus process

Inclusion criteria for the panelists required that at least 2 of the following 3 criteria were met: clinical practice focused on IBD; publication of more than 10 articles on IBD and/or endoscopy as first or senior authors; and expertise in clinical IBD, endoscopy, or IBD pathology. The members of the steering committee all had experience in the preparation of consensus statements or guidelines.

We used the modified Delphi method to guide the preparation of documents.[2] The panel consisted of nationally or internationally recognized IBD, endoscopy, and gastrointestinal

(GI) pathology specialists. The steering committee generated items that reflected questions related to clinical practice into 6 areas of interest based on an extensive literature review: diagnosis, disease monitoring and assessment of treatment response, postoperative care, dysplasia surveillance, interventional IBD, and future perspectives.

The items were first circulated among members of the steering committee. After multiple revisions, the draft was sent to the entire panel for feedback and revised accordingly. Three rounds of voting on the degree of agreement with the items took place via SurveyMonkey (https://surveymonkey.com). The panel members voted anonymously, ranking their agreement with the statements of strongly agree, agree, neutral, disagree, strongly disagree, or do not have expertise. Panel members also provided comments and suggested revisions.

After completing the first and second round surveys, virtual meetings were convened on October 2 and October 10, 2023 for the discussion of statements that had not achieved consensus. An additional survey for interval of surveillance for UC was conducted in early May 2024. A statement was accepted as consensus if >80% of participants voted "agree" or "strongly agree" with the proposed statements (Table 1), excluding "neutral" or "do not have expertise" votes. The number of panelists who voted with "do not have expertise" was not calculated as a denominator. The statements that did not achieve consensus through voting were put in a separate column and were explained in the text (Table 2). The manuscript was reviewed, re-reviewed, and approved by all members of the panel. The preparation and manuscript style follow other recent ASGE consensus statements.[3]

## DIAGNOSIS

CD predominantly affects the distal ileum and/or colon (L1, L2, and L3 disease per the Montreal classification[4]). Colonoscopy with intubation of the terminal ileum is critical for initial diagnostic evaluation for CD.

### Ileocolonoscopy

**Statement 1.** *Ileocolonoscopy is indicated in all patients with chronic diarrhea (>4-6 weeks), persistent bleeding, new or unexplained anemia, weight loss, growth failure in children, or other alarming clinical presentations.*

**Synopsis.** Ileocolonoscopy plays a key role in the diagnosis and differential diagnosis of IBD with other common inflammatory or functional disorders of the lower GI tract, such as irritable bowel syndrome, microscopic colitis, and infectious enterocolitis. The diagnosis of IBD is based on a combined assessment of clinical, endoscopic, histologic, and radiographic features. The terminal ileum is the most common location for CD, whereas at the time of diagnosis the rectum is uniformly affected by UC. Ileocolonoscopy with biopsy sampling is the most reliable tool for the diagnosis of IBD.[5,6] Intubation of the terminal ileum should be considered as one of the quality measurements in the evaluation of IBD, although it can be challenging in some cases.[5,7,8]

**Statement 2.** *Ileocolonoscopy with tissue biopsy sampling is required for the diagnosis of CD and UC.*

**Synopsis.** Most patients with CD have involvement of the terminal ileum. CD is characterized by segmental distribution, transmural inflammation, intestinal strictures, and fistulas and the presence of noncaseating granulomas on tissue biopsy samples in approximately 9% to 67% of patients.[9,10] However, the absence of granulomas on biopsy samples of the GI tract does not exclude the diagnosis of CD. UC is characterized by diffuse inflammation of the rectum with the extension of continuous inflammation proximally in the colon. Inflammation is limited to mucosal and superficial submucosa. On tissue biopsy samples of colonic mucosa, both CD and UC have histologic features of active inflammation (erosions, ulcers, cryptitis, or crypt abscess with infiltration of neutrophils) and chronicity (infiltration of lamina propria by chronic inflammatory cells, crypt distortion or branching, pyloric gland metaplasia, and/or Paneth cell metaplasia). Endoscopically normal-appearing ileum and large-bowel mucosa may also be biopsy sampled.

**Statement 3.** *The use of medications known to induce inflammation in patients with IBD such as nonsteroidal anti-inflammatory drugs should be avoided before diagnostic ileocolonoscopy.*

**Synopsis.** Chronic use of nonsteroidal anti-inflammatory drugs can lead to an enteropathy with erythema, erosions, ulcers, or strictures in the small bowel, particularly the terminal ileum.[11,12] Histologic features of nonsteroidal anti-inflammatory drug enteropathy and CD do overlap. Lymphoplasmacytic inflammation is less frequent in the former.[11]

**Statement 4.** *Adequate cleansing with documentation of bowel preparation scores should be obtained for diagnostic and therapeutic IBD endoscopy.*

**Synopsis.** Adequate bowel preparation is important for ensuring safe and effective diagnostic and therapeutic endoscopy in IBD.[13,14] However, bowel preparation, particularly in those with partial obstruction or IBD, can be challenging. In a study of 730 patients undergoing elective colonoscopy with sodium phosphate–based preparations, endoscopic mucosal lesions, possibly associated with sodium phosphate ingestion, were found in 24 patients (3.3%).[15] Currently, a variety of non-sodium phosphate–based, small-volume bowel cleansing agents are available.

**Statement 5.** *At the index ileocolonoscopy, at least 4 sets of biopsy samples—each set from the terminal ileum, right-sided colon, left-sided colon, and rectum—and additional biopsy samples should be taken from all endoscopically reachable strictures. Index ileocolonoscopy with biopsy sampling should be performed before initiation of medical therapy to avoid "treatment effect," to assess disease extent, and to differentiate CD from UC.*

**Synopsis.** The best time for assessment is at the index ileocolonoscopy before patients are exposed to medical

**TABLE 1. Statements that achieved consensus by the panel**

| Statement no. | Statement | Category | Panel voting results |
|---|---|---|---|
| 1. | Ileocolonoscopy is indicated in all patients with chronic diarrhea (>4-6 wk), persistent bleeding, new or unexplained anemia, weight loss, growth failure in children, or other alarming clinical presentations. | Diagnosis | Strongly agree: 12 (85.7%); agree: 2 (14.3%) |
| 2. | Ileocolonoscopy with tissue biopsy sampling is required for the diagnosis of CD and UC. | | Strongly agree: 12 (85.7%); agree: 2 (14.3%) |
| 3. | The use of medications known to induce inflammation in patients with IBD such as nonsteroidal anti-inflammatory drugs should be avoided before diagnostic ileocolonoscopy. | | Strongly agree: 1 (7.1%); agree: 10 (71.4%); neutral: 2; no expertise: 1 |
| 4. | Adequate cleansing with documentation of bowel preparation scores should be obtained for diagnostic and therapeutic IBD endoscopy. | | Second round voting: strongly agree: 7 (50.0%); agree: 7 (50.0%) |
| 5. | At the index ileocolonoscopy, at least 4 sets of biopsy samples—each set from the terminal ileum, right-sided colon, left-sided colon, and rectum—and additional biopsy samples should be taken from all endoscopically reachable strictures. Index ileocolonoscopy with biopsy sampling should be performed before initiation of medical therapy to avoid "treatment effect," assess disease extent, and differentiate CD from UC. | | Strongly agree: 7 (50.0%); Agree: 7 (50.0%) |
| 6. | Tissue biopsy sampling should be taken from strictures, representative nodular and inflamed mucosa, and the edge of an ulcer. | | Strongly agree: 10 (71.4%); agree: 3 21.4% |
| 7. | We recommend cross-sectional imaging (CT enterography or magnetic resonance enterography) examination around the time of the index ileocolonoscopy to verify the diagnosis, disease extent, and presence of adverse events (eg, stricture and fistula). | | Strongly agree: 9 (64.3%); agree: 3 (21.4%) |
| 8. | Intestinal US, where available, can be considered a tool to assess mucosal and transmural inflammation, stricture, fistula, and abscess as part of the evaluation of diagnosis, disease monitoring, and assessment of treatment response. | | Second round voting: strongly agree: 4 (28.6%); agree: 8 (57.1%) |
| 9. | EGD is recommended for symptomatic adult or all pediatric patients suspected of CD. | | Strongly agree: 8 (57.1%); agree: 3 (21.4%) |
| 10. | Capsule endoscopy after a passed patency capsule may be performed in patients suspected of having small-bowel CD without obstructive symptoms if ileocolonoscopy and/or small-bowel imaging (eg, CT enterography, magnetic resonance enterography, or intestinal US) is not conclusive. | | Strongly agree: 6 (42.9%); agree: 7 (50.0%) |
| 11. | Flexible sigmoidoscopy, colonoscopy, or ileocolonoscopy, along with the assessment of clinical and laboratory biomarkers, is preferred for patients with IBD after the initiation, escalation, or switch of medical therapy to document treatment response. The time interval and endoscopic modality depend on the IBD type, mechanism of action of medications, and response to therapy. | Disease monitoring and assessment of treatment response | Strongly agree: 8 (57.1%); agree: 5 (35.7%) |
| 12. | Disease activity instruments, such as the Simple Endoscopic Score for Crohn's Disease for CD, the Mayo score for UC, and the Rutgeerts score for postoperative CD, should be used for disease assessment. | | Second round voting: strongly agree: 10 (71.4%); agree: 4 (28.6%) |
| 13. | In patients with pelvic or abdominal pouches and persistent symptoms, pouchoscopy should be performed for the assessment of treatment response. | | Second round voting: strongly agree: 7 (50.0%); agree: 7 (50.0%) |
| 14. | Endoscopic mucosal healing, even when histologic healing has not occurred, is sufficient to justify continuing the current therapy. | | Strongly agree: 8 (57.1%); agree: 5 (35.7%) |
| 15. | Flexible sigmoidoscopy with biopsy sampling should be performed to assess the disease activity and evaluate for the potential presence of cytomegalovirus infection in hospitalized (or in ambulatory surgery center) UC patients being treated with steroids, biologics, or small molecules. | | Strongly agree: 7 (50.0%); agree: 6 (42.9%) |

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**TABLE 1. Continued**

| Statement no. | Statement | Category | Panel voting results |
|---|---|---|---|
| 16. | Ileocolonoscopy to monitor disease activity should be performed 6-12 mo after ileocolonic resection and ileocolonic anastomosis and stoma closure (in those with temporary ileostomy) in CD. | Postoperative care | Strongly agree: 11 (78.6%); agree: 3 (21.4%) |
| 17. | Pouchoscopy with biopsy sampling is recommended for the diagnosis and differential diagnosis of pouchitis in symptomatic patients. Pouchoscopy evaluation and description should include anatomic landmarks, such as the prepouch ileum, inlet, pouch body, anastomosis, and cuff. | | Strongly agree: 10 (71.4%); agree: 4 (28.6%) |
| 18. | Disease activity instruments, such as the Pouchitis Disease Activity Index, can be used for the assessment of pouchitis, CD of the pouch, and cuffitis. | | Second round voting: strongly agree: 4 (28.6%); agree: 8 (57.1%) |
| 19. | Ileoscopy through the stoma is recommended before stoma closure in CD patients with temporary fecal diversion; the ileoscope should be introduced beyond the level of the fascia. | | Strongly agree: 6 (42.9%); agree: 6 (42.9%) |
| 20. | Surveillance colonoscopy every 1-5 y is recommended in patients with Crohn's colitis or UC 8-10 y after diagnosis with disease extending beyond the rectum. Surveillance interval is determined by patient-specific factors (prior severity of colonic inflammation and history of dysplasia, family history of colorectal cancer, primary sclerosing cholangitis) and frequency, quality, and modality of prior surveillance examinations. | Surveillance | Third round voting: strongly agree: 2 (13.3%); agree: 12 (80%); disagree: 1 (.06%) |
| 21. | All UC-associated strictures in the colon or rectum should be biopsy sampled at least once per year to evaluate for the presence of neoplasia. | | Strongly agree: 6 (42.9%); agree: 6 (42.9%) |
| 22. | All patients with primary sclerosing cholangitis should undergo a diagnostic colonoscopy for the evaluation of UC at the time of diagnosis of primary sclerosing cholangitis; all patients with UC, CD involving the large bowel, and primary sclerosing cholangitis should have yearly surveillance colonoscopy regardless of the extent and duration of IBD with enhanced imaging technology (narrow-band imaging or chromoendoscopy) with mucosal biopsy sampling. | | Strongly agree: 8 (57.1%); agree: 4 (28.6%) |
| 23. | Biopsy samples of CD-associated colonic, rectal, and anorectal strictures should be taken at the index colonoscopy and every 1-3 y afterward to rule out malignancy. | | Strongly agree: 6 (42.9%); agree: (57.1%) |
| 24. | Surveillance biopsy sampling should be performed every 1-3 y for patients with diverted large bowel and duration of UC or CD for more than 8-10 y. | | Strongly agree: 4 (28.6%); agree: 9 (64.3%) |
| 25. | Yearly surveillance pouchoscopy should be done for IBD patients at high risk for dysplasia, that is, those with precolectomy colitis-associated neoplasia. | | Strongly agree: 9 (64.3%); agree: 5 (35.7%) |
| 26. | Surveillance pouchoscopy every 1-3 y should be performed in patients with IBD at moderate risk for dysplasia (eg, primary sclerosing cholangitis, chronic pouchitis, CD of the pouch, chronic cuffitis, and family history of colorectal cancer. | | Strongly agree: 9 (64.3%); agree: 3 (21.4%) |
| 27. | Surveillance pouchoscopy should be performed every 3 y in IBD patients at average risk for dysplasia. | | Strongly agree: 3 (21.4%); agree: 9 (64.3%) |
| 28. | High-definition white-light endoscopy or image-enhanced endoscopy (such as virtual or dye-based chromoendoscopy) with targeted or random biopsy sampling should be used for surveillance colonoscopy. | | Second round voting: strongly agree: 7 (50.0%); agree: 6 (42.9%) |
| 29. | High-definition white-light endoscopy or image-enhanced endoscopy (eg, virtual or dye-based chromoendoscopy) with targeted and random biopsy sampling should be used for surveillance colonoscopy in high-risk patients, such as those with primary sclerosing cholangitis. | | Second round voting: strongly agree: 8 (57.1%); agree: 5 (35.7%) |

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**TABLE 1. Continued**

| Statement no. | Statement | Category | Panel voting results |
|---|---|---|---|
| 30. | Intralesional injection of long-acting steroids is not recommended before, during, or after endoscopic balloon dilation for strictures. | Interventional IBD | Strongly agree: 7 (50.0%); agree: 5 (35.7%) |
| 31. | Endoscopic balloon dilation of deeply ulcerated strictures should be avoided. | | Strongly agree: 7 (50.0%); agree: 5 (35.7%) |
| 32. | The long-term success of endoscopic stricture therapy is measured by reintervention-free survival and surgery-free survival. | | Strongly agree: 5 (35.7%); agree: 9 (64.3%) |
| 33. | Large (>1 cm) inflammatory polyps may be removed to reduce the symptoms of bleeding, obstruction, and anemia, even though the risk of dysplasia is low. | | Strongly agree: 3 (21.4%); agree: 9 (64.3%) |
| 34. | Endoscopic stents should not be used in the treatment of benign disease–associated or anastomotic strictures in pre- or postoperative CD or UC. | | Strongly agree: 7 (50.0%); agree: 4 (28.6%) |
| 35. | Surgical intervention should occur for CD strictures, especially long (>4-5 cm) or complex (eg, fistula and/or abscess-associated strictures, those refractory to previous intervention, or recurrent [requiring endoscopic intervention more often than every 3-6 mo]) strictures after previously successful interventions. | | Strongly agree: 10 (71.4%); agree: 4 (28.6%) |
| 36. | Polypectomy, EMR, or endoscopic submucosal dissection may be performed on polypoid or raised, liftable dysplastic lesions with a clear, well-defined border. | | Strongly agree: 8 (57.1%); agree: 6 (42.9%) |
| 37. | The exploration of artificial intelligence in the diagnosis and surveillance of IBD should continue. | Future perspectives | Strongly agree: 7 (50.0%); agree: 6 (42.9%) |
| 38. | Applications of novel endoscopic devices in interventional IBD (eg, drug-coated balloons, scope-tip devices, suturing devices, and endoscopic delivery systems for cell therapy) should be explored. | | Strongly agree: 6 (42.9%); agree: 8 (57.1%) |

*CD*, Crohn's disease; *IBD*, inflammatory bowel disease; *UC*, ulcerative colitis.

therapy. Medical treatment of UC can result in a patchy distribution of inflammation in one-third of patients.[16] In adult patients with CD, disease location and distribution appear to remain relatively stable, whereas disease extent and distribution of UC often change.[17]

In healthy people, histologic features of enteric and colonic mucosa vary by location. Enteric biopsy samples typically show mucosa with architectural regularity characterized by evenly sized and spaced villi (ileum) and crypts (ileum, colon, and rectum) with minimal crypt distortion. Scattered lymphocytes are present in the epithelium, and chronic inflammatory cells (plasma cells, lymphocytes, eosinophils) are present in the lamina propria. The density of the chronic inflammatory infiltrate decreases from the cecum to the rectum in healthy people. Extravascular neutrophils are not normally seen in the ileum and colon, and their presence indicates active inflammation. In the setting of IBD, expansion of the mucosa by chronic inflammatory cells and crypt architectural distortion (in forms of villous blunting in the ileum, branching and/or shortening of crypts, villiform appearance of colonic surface epithelium) are seen along with activity as measured by neutrophilic infiltrate with cryptitis or crypt abscess.

Paneth cell metaplasia, defined as the presence of Paneth cells found distal to the splenic flexure, is a histologic sign of chronic mucosal injury and can be seen in CD or UC. In non-IBD patients, including infectious colitis, ischemic colitis, and mucosal prolapse, Paneth cell metaplasia was rarely (0%-1.9%) seen in the distal large bowel.[18]

Pyloric gland metaplasia is a common finding in intestinal specimens of patients with CD.[19] It can occur in the colonic mucosa in patients with IBD (both CD and UC), but the frequency is much lower than in the ileal mucosa in IBD. In young patients without a history of prior intestinal surgery, the presence of pyloric gland metaplasia accompanied by severe active inflammation or crypt distortion in small-bowel mucosa is strongly indicative of CD.[17] Segmental colitis associated with diverticulosis or diverticular colitis typically affects the sigmoid colon and mimics endoscopic and histologic features in CD or UC. Segmental colitis associated with diverticulosis should spare the rectum; therefore, biopsy sampling of the rectum is important for the diagnosis.[20,21]

Another mimic of IBD is solitary rectal ulcer syndrome, rectal prolapse, or rectocele, which may present with mucosal inflammation at the distal rectum, particularly at the anterior wall. To distinguish from IBD, the rectal biopsy sample is preferably taken from the anterior wall.

Various histology score instruments (eg, the Geboes score,[22] Nancy Histological Index,[23] and Robarts Histopathology Index[24]) have been developed for clinical trials and practice. Accurate assessment of the distribution and characteristics of macroscopic and microscopic inflammation is

**TABLE 2. Statements that did not achieve consensus by the panel**

| No. | Statements |
|---|---|
| 1 | The use of agents known to induce inflammation in patients with IBD such as nonsteroidal anti-inflammatory drugs should be held for ≥4 wk before performing ileocolonoscopy in known IBD. |
| 2 | Sodium phosphate–based bowel preparation agents for lower GI endoscopy should be avoided. PEG-based bowel preparation is preferred. |
| 3 | A jumbo biopsy sample is preferred for primary or anastomotic strictures in patients at risk for dysplasia, such as those with a long duration of IBD, concurrent primary sclerosing cholangitis, or bowel resection for bowel neoplasia. |
| 4 | We encourage the onsite use of intestinal US, if available, to assess mucosal and transmural inflammation, stricture, fistula, abscess, and lymphadenopathy as part of the evaluation of diagnosis, disease monitoring, and assessment of treatment response. |
| 5 | Diagnostic or therapeutic push enteroscopy or device-assisted enteroscopy, if available, is recommended in patients suspected of small-bowel CD when ileocolonoscopy and small-bowel imaging are not feasible or inconclusive. |
| 6 | Ileoscopy through the stoma every 1-3 y is recommended in CD patients with permanent fecal diversion. |
| 7 | Periodic surveillance for glandular or squamous cell neoplasia with modalities such as endoscopic biopsy sampling, anal smears, and examination under anesthesia should be considered for patients with perianal CD, especially in those at risk (eg, longstanding CD, human papillomavirus, or HIV). |
| 8 | Direct tissue biopsy sampling may be avoided on lesions highly suspicious of dysplasia to reduce the difficulty of anticipated EMR or endoscopic submucosal dissection in patients who will undergo these procedures shortly. However, biopsy samples should be taken and a tattoo should be injected from adjacent mucosa. |
| 9 | Endoscopic evaluation of the surgical strictureplasty site should include the inlet, lumen, and outlet. |
| 10 | A bypassed segment of the bowel, such as a strictured duodenum with gastrojejunostomy, should be endoscopically surveyed every 3 y for malignancy. |
| 11 | Yearly surveillance biopsy sampling is recommended to exclude glandular or squamous cell dysplasia in patients with chronic (>5 y) perianal disease in CD. |
| 12 | Image-enhanced endoscopy with targeted or random biopsy sampling, such as virtual or dye-based chromoendoscopy, is recommended for surveillance colonoscopy. |
| 13 | Endoscopic stricture treatment may be performed in all IBD-associated strictures, regardless of the presence or absence of symptoms. |
| 14 | Technical success (short-term success) of endoscopic treatment of stricture is measured by the passage of a pediatric colonoscope (or upper endoscope) with or without resistance. |
| 15 | We recommend the establishment of a multidisciplinary team, consisting of an IBD specialist, advanced endoscopist, colorectal surgeon, anesthesiologist, GI radiologist, and GI pathologist, for interventional IBD service. |
| 16 | We recommend that interventional IBD training should be incorporated into the advanced endoscopy or IBD fellowship (postgraduate year 7) curriculum. |
| 17 | We recommend the establishment of an interventional IBD Center of Excellence with credentialing from American Society for Gastrointestinal Endoscopy to ensure quality outcomes. Criteria for the Center of Excellence include available interventional IBD service (eg, endoscopic balloon dilation, stricturotomy, EMR, and endoscopic submucosal dissection of the lower GI tract) and a multidisciplinary team, consisting of an IBD specialist, advanced endoscopist, colorectal surgeon, anesthesiologist, GI radiologist, and GI pathologist. |

*CD*, Crohn's disease; *IBD*, inflammatory bowel disease.

critical for assessing whether a patient has a diagnosis of Crohn's colitis or UC.

**Statement 6.** *A tissue biopsy sample should be taken from strictures, representative nodular and inflamed mucosa, and the edge of an ulcer.*

**Synopsis.** In patients with IBD who have intestinal strictures, nodular mucosa, inflamed mucosa, mucosal ulceration, or other significant pathology, the diagnostic yield parallels the number of biopsy samples taken. A recent study found that in the setting of active colitis, 3 biopsy samples per region of interest was the minimum number required to overcome microscopic heterogeneity and ensure accurate histologic grading.[25]

**Statement 7.** *We recommend cross-sectional imaging (CT enterography [CTE] or magnetic resonance enterography [MRE]) examination around the time of the index ileocolonoscopy to verify the diagnosis, assess disease extent, and evaluate for adverse events (such as stricture and fistula).*

**Synopsis.** Most patients with CD or UC have some form of cross-sectional imaging of the abdomen and pelvis before, at, or after diagnosis. This is particular true in patients with CD. To overcome the limitation of ileocolonoscopy in the assessment of disease of the proximal small bowel, extraluminal/mural, or extramural lesions, cross-sectional imaging such as CTE or MRE is recommended. CTE or MRE helps to further characterize disease distribution, severity, disease phenotype,

and adverse events of IBD and therefore is recommended at the time of diagnosis and suspected adverse events. Intestinal US (IUS) is emerging as a feasible alternative to CTE or MRE.[26,27]

**Statement 8.** *IUS, where available, can be considered a tool to assess mucosal and transmural inflammation, stricture, fistula, and abscess as part of the evaluation of diagnosis, disease monitoring, and assessment of treatment response.*

**Synopsis.** The application of IUS in the clinical practice of CD and UC is evolving.[28] Although equipment procurement and training of ultrasonographers can be a challenge, we expect an expansion of onsite IUS in diagnosis, disease monitoring, and assessment of treatment response. Large multicenter prospective trials are needed to fully understand how to incorporate IUS in the routine management of patients with IBD.[29] IUS is particularly useful in monitoring disease activity in women during pregnancy.[30]

### EGD

**Statement 9.** *EGD is recommended for symptomatic adults and all pediatric patients suspected of CD.*

**Synopsis.** CD can involve any part of the GI tract, including the esophagus, stomach, and duodenum, as L4 lesions in the Montreal classification.[4] The natural history and disease course of pediatric patients with CD can be different from adult patients. For example, upper GI CD is more common in pediatric patients than in their adult counterparts. Therefore, EGD has a higher yield of CD in children than in adults.[31] In addition, EGD should be performed in adult patients suspected of CD who have upper GI symptoms, such as dyspepsia, dysphagia, and epigastric pain, to increase diagnostic yield and to identify alternative diagnoses.

### Video capsule endoscopy

**Statement 10.** *Capsule endoscopy after a passed patency capsule may be performed in patients suspected of having small bowel CD without obstructive symptoms if ileocolonoscopy and/or small bowel imaging (eg, CTE, MRE, or IUS) is not conclusive.*

**Synopsis.** The application of video capsule endoscopy (VCE) in the diagnosis of CD of the small bowel is limited because of the inherent inability to take tissue samples, presence of contraindications such as intestinal strictures, risk of a retained capsule, and readily available cross-sectional imaging and deep enteroscopy. Some clinicians believe that VCE may provide a highly sensitive test to evaluate early mucosal changes, particularly in CD. A meta-analysis of 5 observational studies including 142 patients showed that mucosal healing on VCE score was found to be significantly associated with improved outcomes after a follow-up of 12 weeks to 24 months.[32]

Concern about the risk of capsule retention exists. A pooled analysis showed that retention rates were 3.32% (95% confidence interval [CI], 2.62-4.2) in an overall CD population, 2.35% (95% CI, 1.31-4.19) in suspected CD, and 4.63% (95% CI, 3.42-6.25) in established CD.[33] The capsule retention rates in adult CD and pediatric CD were reported to be 3.49% (95% CI, 2.73-4.46) and 1.64% [95% CI, .68-3.89), respectively.[29] In patients with established CD, the retention rates in those with the use of pre-VCE or cross-sectional imaging were 2.88% (95% CI, 1.74-4.74) and 2.32% (95% CI, .87-6.03), respectively.[29] As a safety precaution, a patency capsule or CT and/or magnetic resonance imaging before VCE may be used, particularly in those with obstructive symptoms, inconclusive abdominal imaging, or a history of bowel surgery.

## DISEASE MONITORING AND ASSESSMENT OF TREATMENT RESPONSE

A poor correlation exists between symptom scores and the degree of endoscopic inflammation in IBD, particularly CD. Endoscopic evaluation provides the most reliable and objective measurement of disease activity. Endoscopic mucosal healing has become a treatment target for medical therapy in IBD.[34]

**Statement 11.** *Flexible sigmoidoscopy, colonoscopy, or ileocolonoscopy, along with the assessment of clinical and laboratory biomarkers, is preferred for patients with IBD after the initiation, escalation, or switch of medical therapy to document treatment response. The time interval and endoscopic modality depend on the IBD type, mechanism of action of medications, and response to therapy.*

**Synopsis.** Growing evidence suggests that histologic healing or histologic improvement beyond endoscopic remission is associated with favorable long-term outcomes, although histologic healing has not been set to be a therapeutic target in clinical guidelines.[30] Documentation of endoscopic mucosal healing with or without histologic healing requires proper endoscopic evaluation.

**Statement 12.** *Disease activity instruments, such as the Simple Endoscopic Score for Crohn's Disease for CD, Mayo score for UC, and Rutgeerts score for postoperative CD, should be used for disease assessment.*

**Synopsis.** Commonly used disease activity indices are the 44-point Crohn's Disease Endoscopic Index of Severity,[35] Simple Endoscopic Score for Crohn's Disease (Fig. 1),[36] Truelove-Witts score for UC,[37] Mayo score for UC (Fig. 2),[38] and Rutgeerts score for postoperative CD (Fig. 3),[39] including in patients with an ileostomy.[40] These disease activity instruments may be incorporated into quality measurements. The GI Quality Improvement Consortium registry is a voluntary nationwide quality improvement registry that was established in 2010 in a collaboration between the ASGE and American College of Gastroenterology and advocates the use of an endoscopic score in all patients with IBD (https://giquic.org/).

**Statement 13.** *In patients with pelvic or abdominal pouches and persistent symptoms, pouchoscopy should be performed for the assessment of treatment response.*

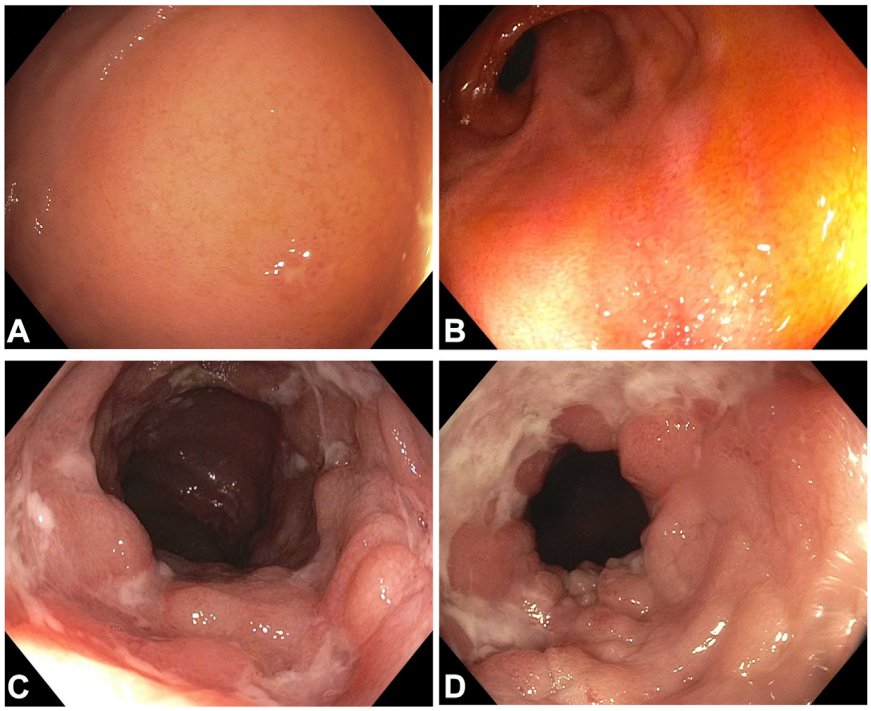


**Figure 1.** Endoscopic scoring of mucosal inflammation in Crohn's disease using the Simple Endoscopy Score for Crohn's Disease (SES-CD). **A,** SES-CD ulcer score of 0, no erosions or ulcers. **B,** SES-CD ulcer score of 1: aphthous ulcers .1 to .5 cm. **C,** SES-CD ulcer score of 2, large ulcers .5 cm to 2 cm. **D,** SES-CD ulcer score of 3, very large ulcers >2 cm.

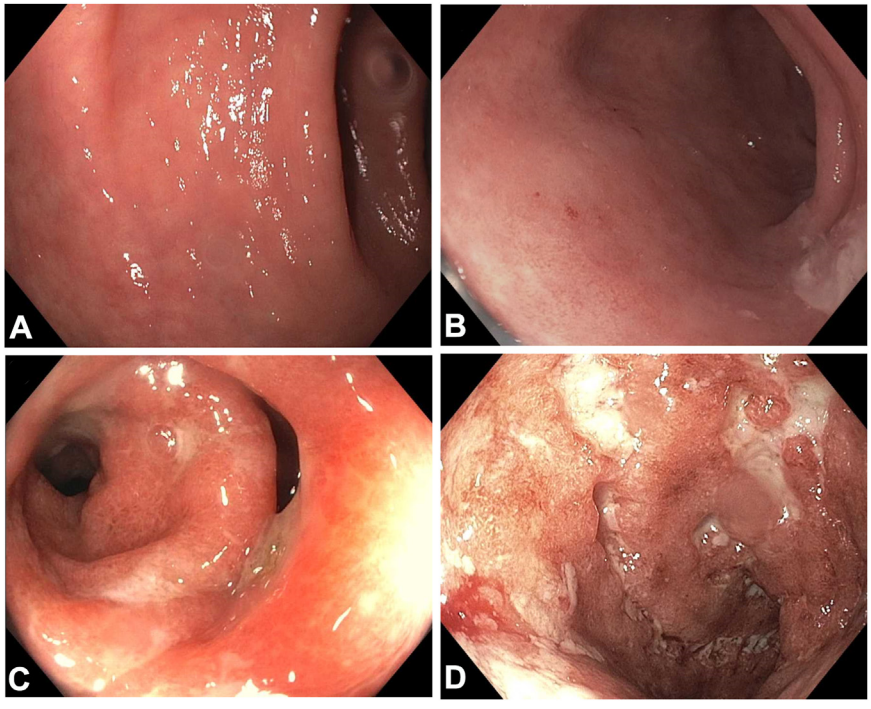


**Figure 2.** Endoscopic scoring of mucosal inflammation in ulcerative colitis using the Mayo Endoscopy Subscores (MES). **A,** MES of 0, no friability or granularity, intact vascular pattern. **B,** MES of 1, erythema, diminished vascular markings, and mild granularity. **C,** MES of 2, marked erythema, absent vascular marking, granularity, no ulceration. **D,** MES of 3, marked erythema, absent vascular markings, granularity, friability, spontaneous bleeding in the lumen, and ulcerations.

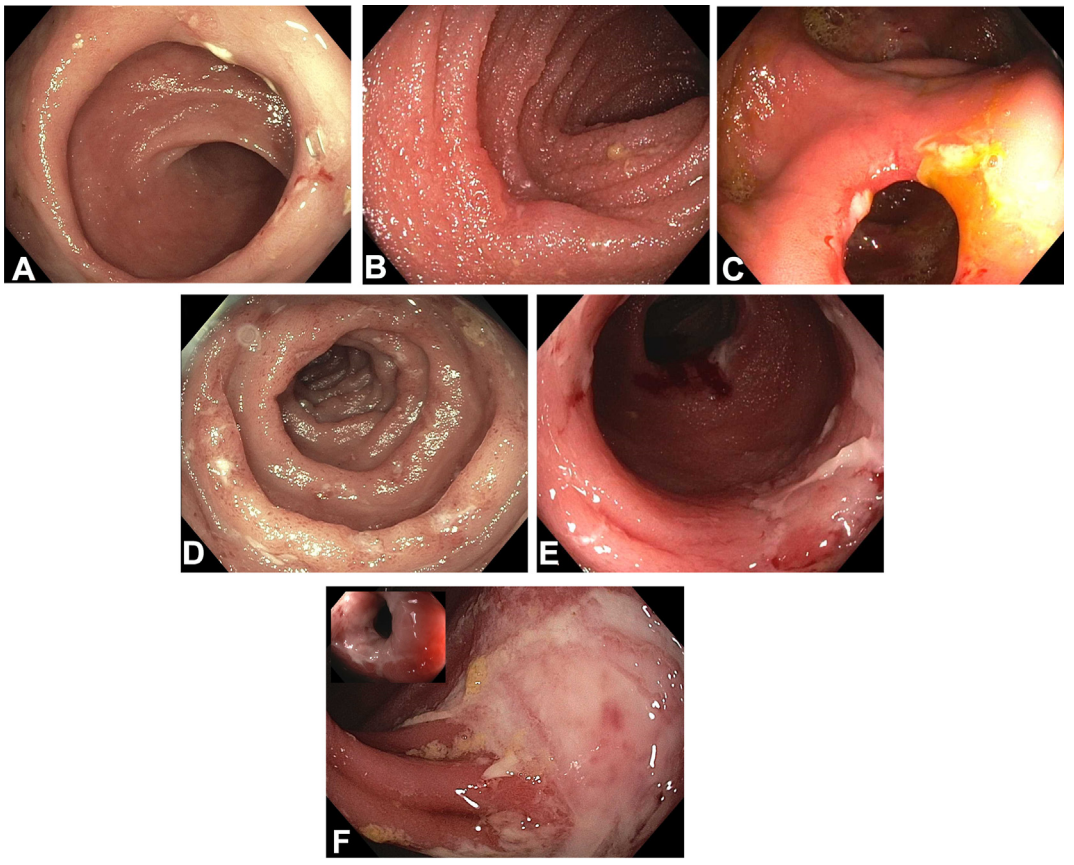


**Figure 3.** Endoscopic scoring of postoperative recurrence of Crohn's disease in the neoterminal ileum using the modified Rutgeerts score (RS). **A,** RS of i,0, no lesions. **B,** RS of i,1, <5 aphthous lesions; **C,** RS of i,2a, erosions in the ileocolonic anastomosis. **D,** RS of i,2b, >5 aphthous lesions with normal mucosa in between the lesions. **E,** RS of i,3, diffuse aphthous ileitis with diffusely inflamed mucosa. **F,** RS of i,4, diffuse inflammation with already large ulcers, nodules, and/or narrowing (in box).

**Synopsis.** Most patients with acute pouchitis respond favorably to antibiotic therapy. Some patients may develop chronic antibiotic-dependent pouchitis or chronic antibiotic-refractory pouchitis. Management of chronic antibiotic-refractory pouchitis can be challenging, and this phenotype is considered 1 of the 5 difficult-to-treat IBD conditions, according to a consensus statement of an expert panel.[41] Closely monitoring of disease activity and treatment response is important in those patients with chronic pouchitis who are receiving medical therapy. Elevated fecal calprotectin and lactoferrin levels correlated with endoscopic and histologic inflammation of the pouch or predictors of pouchitis.[42-45] However, these fecal markers cannot further differentiate inflammatory disorders of the pouch, such as acute or chronic pouchitis, CD or CD-like condition of the pouch, or cuffitis. Additionally, incomplete mucosal healing may occur with normalization of the fecal calprotectin. Assessment for histologic improvement of inflammation might be helpful to assess treatment response.[46]

**Statement 14.** *Endoscopic mucosal healing, even when histologic healing has not occurred, is sufficient to justify continuing the current therapy.*

**Synopsis.** Investigators have proposed that histologic scores, such as the Geboes score, Nancy Histological Index, and Robarts Histopathology Index, should be part of the core outcomes of clinical trials.[20-22,47] Although histologic healing is considered a potential future treatment goal, in current clinical practice, there is no consensus in the literature regarding its implementation as a treatment target. Endoscopic remission in the absence of partial or complete histologic remission is not viewed as a treatment failure. Laboratory markers, including commonly used fecal calprotectin, may serve as a surrogate marker for disease monitoring and assessment of mucosal healing in CD[48] or UC.[49] Endoscopy and histology should be combined with clinical, laboratory, and radiographic assessments for disease monitoring.

**Statement 15.** *Flexible sigmoidoscopy with biopsy sampling should be performed to assess the disease activity and evaluate for the potential presence of cytomegalovirus (CMV) infection in hospitalized (or in acute ambulatory surgical center) UC patients being treated with steroids, biologics, or small molecules.*

**Synopsis.** An association exists between CMV infection and exacerbation or refractoriness of UC. Antiviral therapy in high-grade CMV infection favorably affects disease outcomes, such as the requirement of colectomy.[50] The most efficacious and safest way to document CMV infection is a

tissue biopsy sample from the colon with or without CMV-DNA in the blood. However, false-negative results are possible either on biopsy sampling or on testing for CMV-DNA in the blood.[44,51]

The yield of biopsy samples depends on the density of viral inclusions in the tissue and the location. To enhance the yield, the endoscopist may take the biopsy sample from the base and edge of the ulcer, obtaining an adequate number of biopsy samples at different locations of the colon. McCurdy et al[52] recommended that 11 biopsy samples using flexible sigmoidoscopy should be taken for UC and 16 biopsy samples using colonoscopy should be taken for CD,[52] although this is not common in clinical practice.

An immunostain for the CMV antigen can be performed to enhance the detection of CMV-infected cells when there is strong clinical suspicion and equivocal CMV-infected cells on hematoxylin & eosin staining. In immunostain-positive cases, the density of positive CMV cells per biopsy section should be reported by the pathologist. A study by Clos-Parals et al[53] found, on multivariate analysis, a significant association with colectomy in UC patients with active disease and >2 immunohistochemistry-positive CMV cells per biopsy sample. In cases with equivocal CMV inclusions on immunohistochemistry, testing additional concurrent intestinal biopsy samples can be performed to increase diagnostic yield. CMV testing by immunohistochemistry or polymerase chain reaction from the tissue has a high sensitivity,[54] but the significance of positive polymerase chain reaction only without concurrent positive histology is unclear.

## POSTOPERATIVE MANAGEMENT

Surgery for CD or UC involves resection, anastomosis, or reconstruction. Attempts have been made to standardize the practice of endoscopic evaluation of the postoperative GI tract in patients with surgically altered anatomy.[55] Endoscopic evaluation of a surgically altered gut should include the evaluation and description of anatomic landmarks in the healthy or diseased state.

### Ileocolonic resection

**Statement 16.** *An ileocolonoscopy to monitor disease activity should be performed 6 to 12 months after ileocolonic resection and ileocolonic anastomosis and stoma closure (in those with temporary ileostomy) in CD.*

**Synopsis.** The Rutgeerts score is commonly used for the monitoring of postoperative recurrence of CD,[35] although the Simple Endoscopic Score for Crohn's Disease was also shown to have predictive value.[32,56] Close postoperative monitoring with ileocolonoscopy appears to be associated with better clinical outcomes. In a randomized trial, intensified postoperative endoscopic monitoring (such as ileocolonoscopic evaluation as early as 6 months after ileocolonic resection for CD) was shown to yield better clinical outcomes than standard practice, which was defined as an 18-month ileocolonoscopy after surgery without a 6-month ileocolonoscopy.[57] After the index ileocolonoscopy, the interval for ileocolonoscopy for postoperative CD assessment is not certain. However, an interval of 1 to 3 years is reasonable, depending on disease activity, fecal calprotectin, and whether IUS was used.

### Ileal pouch

**Statement 17.** *Pouchoscopy with biopsy sampling is recommended for the diagnosis and differential diagnosis of pouchitis in symptomatic patients. Pouchoscopy evaluation and description should include anatomic landmarks, such as the prepouch ileum, inlet, pouch body, anastomosis, and cuff.*

**Synopsis.** Pouchoscopy is a main diagnostic modality for inflammatory, structural, and neoplastic disorders of the ileal pouch (Fig. 4).[58,59] Pouchoscopy is more reliable than symptomatic and histologic evaluation for the diagnosis and differential diagnosis (from functional disorders of the pouch, such as irritable pouch syndrome) of pouchitis.[60-62] The Pouchitis Disease Activity Index and its variants may be used to quantify endoscopic inflammation of the prepouch ileum, pouch body, and cuff.[63-65] Although scoring endoscopic inflammation varies greatly among investigators, documentation of ulceration and ulcerated surface area in the pouch body appears to be consistent.[66]

Evaluation and description of anastomotic landmarks of the ileal pouch and their diseased status are important for the diagnosis of structural, inflammatory, and neoplastic disorders.[47] Some investigators have used these anatomic locations to classify inflammatory disorders of the pouch.[62,67] Biopsy sampling of the prepouch ileum, pouch body, and rectal cuff can help assess pouchitis severity and provides clues to other etiologies such as superimposed CMV infection,[68,69] ischemia,[41,70] CD of the pouch,[71] and prolapse.

**Statement 18.** *Disease activity instruments, such as the Pouchitis Disease Activity Index, can be used for the assessment of pouchitis, CD of the pouch, and cuffitis.*

**Synopsis.** Pouchoscopy and calculation of the Pouchitis Disease Activity Index[63,72] and its modified form[73] have been used for diagnosis, disease monitoring, and assessment of treatment response in clinical trials.[74] The Pouchitis Disease Activity Index is variably used in clinical practice.

### Ileostomy

**Statement 19.** *Ileoscopy through the stoma is recommended before stoma closure in CD patients with temporary fecal diversion; the ileoscope should be introduced beyond the level of the fascia.*

**Synopsis.** The segment between the stoma and fascia level of the distal bowel may have ulcers, strictures, or fistula that can result from mechanical or ischemic factors, not necessarily reflecting recurrent CD. Therefore, the ileoscope should be advanced beyond the fascia level.

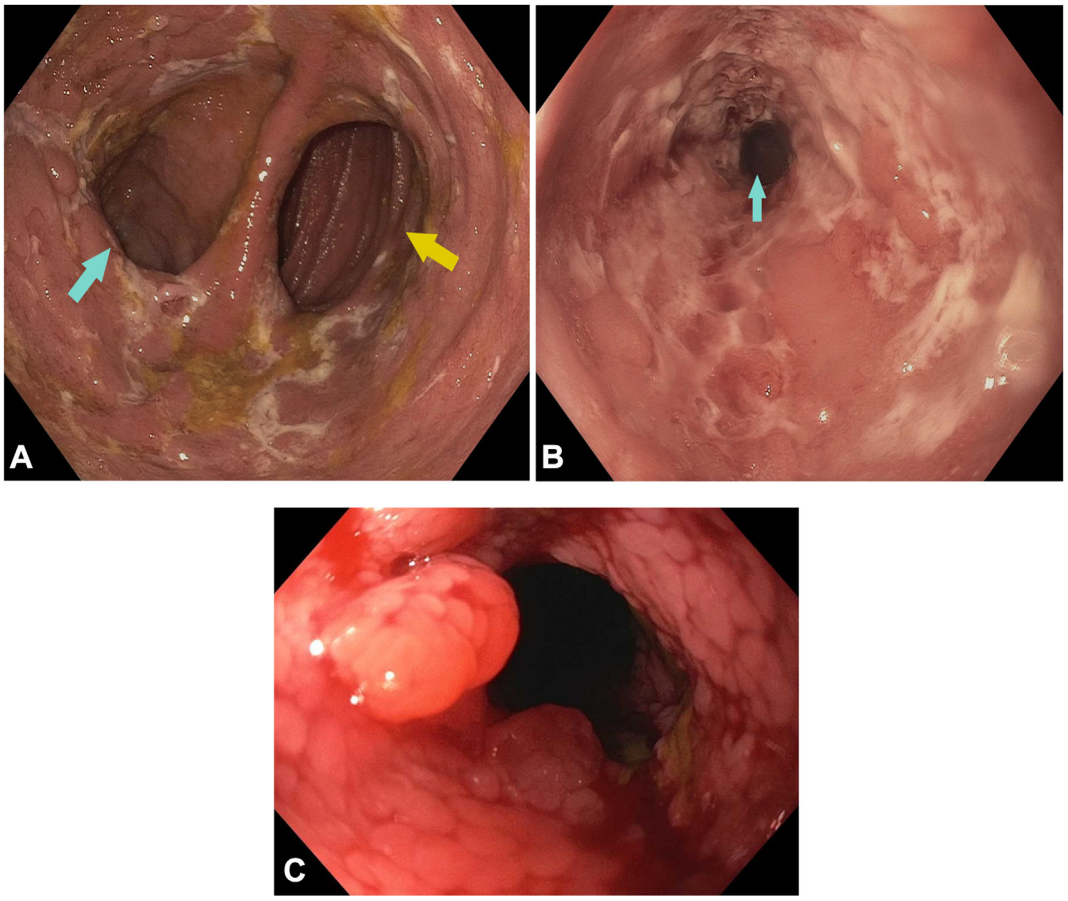


**Figure 4.** Pouchitis and other inflammatory disorders of the pouch. **A,** Pouchitis with diffuse small and large ulcers. The *blue arrow* shows the tip of the "J" and the *yellow arrow* demonstrates the pouch inlet. **B,** Cuffitis. The *blue arrow* illustrates a pouch-anal anastomosis proximally. **C,** Crohn's disease of the pouch with inflammation and stricture in the prepouch ileum.

Alternatively, fecal calprotectin from the ileostomy output may be used to predict small-bowel disease in patients with ileostomy.[75]

## SURVEILLANCE

Patients with UC and Crohn's colitis are at risk for the development of colorectal cancer (CRC). CD- or UC-associated adenocarcinoma has a worse prognosis than sporadic CRC because of its association with a higher incidence of poorly differentiated and laterally spreading cancer. It can often be found within primary or anastomotic strictures, although it is more commonly found in primary strictures. Endoscopic surveillance is required for early detection and management of colitis-associated neoplasia (CAN). Patients with a long duration of disease, persistent endoscopic or histologic inflammation,[76-79] or presence of a prior history of CAN[80,81] or primary sclerosing cholangitis (PSC)[82-84] carry an even higher risk for CAN. The practice pattern of surveillance for dysplasia in IBD has been evolving for the last 3 decades, corresponding with a better understanding of the disease course, carcinogenetic pathways, advances in medical therapy, and endoscopic technology for CAN detection and treatment.[85-92] Surveillance endoscopy is better performed while the disease is in endoscopic remission. Endoscopically visible lesions should be further described as to their site, size, shape (Paris classification), and surrounding inflammation.[93]

**Statement 20.** *Surveillance colonoscopy every 1 to 5 years is recommended in patients with Crohn's colitis or UC 8 to 10 years after diagnosis with disease extending beyond the rectum. The surveillance interval is determined by patient-specific factors (prior severity of colonic inflammation and history of dysplasia, family history of CRC, PSC) and frequency, quality, and modality of prior surveillance examinations.*

**Synopsis.** Various guidelines or position statements were proposed by professional societies or task-based consensus panels. For patients with left-sided or extensive UC for more than 8 to 10 years, a surveillance colonoscopy every 1 to 3 years should be performed, as suggested by a guideline from American College of Gastroenterology.[91] The natural history and carcinogenesis pathways of colitis-associated neoplasia varies and complex.[94] In a subset of patients with long-term disease in endoscopic and

histologic remission and a series of negative surveillance colonoscopy, the surveillance interval may be extended up to every 5 years, as suggested by guidelines or expert reviews from the American Gastroenterological Association,[88] European Crohn's and Colitis Organisation,[95] and British Society of Gastroenterology.[96] The risk for CAN in Crohn's colitis is comparable with that in UC.[97-102]

The historical Seattle surveillance protocol of taking 4-quadrant random biopsy samples from each 10-cm interval including 5 random biopsy samples from the rectum with a minimum of 33 pieces[103] is being phased out in many IBD centers in the average-risk patients. But random biopsies can be done at the initial surveillance as a baseline for the staged biopsy. Image-enhanced endoscopy with targeted biopsy sampling reduces the total number of biopsy samples with improved[78,104] or comparable sensitivity[105,106] with the random biopsy sampling protocol.

**Statement 21.** *All UC-associated strictures in the colon or rectum should be biopsy sampled at least once per year to evaluate for the presence of neoplasia.*

**Synopsis.** UC involves the epithelia, lamina propria, muscularis mucosae, and superficial submucosa. Strictures in UC are generally considered malignant until proven otherwise. On the other hand, submucosal fibrosis is not uncommon, and benign strictures can occur in patients with UC.[107] UC-associated strictures, a major risk factor for CAN,[108] should be regularly biopsy sampled to rule out neoplasia.

**Statement 22.** *All patients with PSC should undergo a diagnostic colonoscopy for the evaluation of UC at the time of diagnosis of PSC. All patients with UC, CD involving the large bowel, and PSC should have yearly surveillance colonoscopy regardless of the extent and duration of IBD with enhanced imaging technology (narrow-band imaging or chromoendoscopy) with mucosal biopsy sampling.*

**Synopsis.** The presence of PSC in patients with IBD involving the large bowel conveys additional risk for the development[109] and poor prognosis of CAN.[110] For high-risk patients including those with PSC, tubular colon, and a prior history of endoscopically invisible dysplasia, the Seattle protocol is likely have added value in addition to targeted biopsy sampling.[111] The interval between the first IBD-negative colonoscopy and subsequent diagnostic colonoscopies varies among clinicians.

**Statement 23.** *Biopsy samples of CD-associated colonic, rectal, and anorectal strictures should be taken at the index colonoscopy and every 1 to 3 years afterward to rule out malignancy.*

**Synopsis.** Crohn's colitis appears to carry a comparable risk for the development of CAN as UC.[100,112-114] Colonic, rectal, or anorectal strictures in the setting of CD may obscure dysplasia or invasive carcinoma. Therefore, surveillance colonoscopy with biopsy sampling should be performed every 1 to 3 years.

**Statement 24.** *Surveillance biopsy sampling should be performed every 1 to 3 years for patients with diverted large bowel and a duration of UC or CD for more than 8 to 10 years.*

**Synopsis.** Available studies on the risk and surveillance of dysplasia in patients with long-term diverted bowel are limited. In a retrospective study of 154 patients with CD or UC and 754 diverted patient-years and 1984 patient-years of disease, 2 cases of diverted colorectal dysplasia (1 case of CD and 1 case of UC) and 1 case of diverted CRC (UC) were observed.[115] In a meta-analysis of 37 studies in which 1211 IBD patients were included (613 UC [50.6%], 524 CD [43.3%], 66 IBD-unclassified [5.4%], and 8 unspecified [.7%] patients), the estimated incidence of cancer in the rectal stump varied from 3.9 to 4.5 per 1000 diverted patient-years.[116] Routine surveillance endoscopy is recommended if completion proctectomy is not feasible according to the Global Interventional IBD Group[117] and other investigators.[104]

**Statement 25.** *Yearly surveillance pouchoscopy should be performed for IBD patients at high risk for dysplasia, that is, those with precolectomy CAN.*

**Synopsis.** Despite proctocolectomy, patients with ileal pouch-anal anastomosis still carry a risk for CAN. The established risk factor for pouch neoplasia is precolectomy CAN.[118,119] The prognosis of pouch cancer is poor. For example, in a case series of 14 patients with pouch cancer, 6 (42.9%) died after a median follow-up of 2.1 years (range, .6-5.2).[120] For patients with UC and ileal pouch-anal anastomosis and precolectomy CAN, yearly surveillance pouchoscopy is recommended.[84] Ileal pouch-anal anastomosis–associated neoplasia mostly involves the cuff or anal transition zone.[106-108] The surveillance biopsy sample should include the cuff and anal transition zone.

**Statement 26.** *Surveillance pouchoscopy every 1 to 3 years should be performed in patients with IBD at moderate risk for dysplasia (eg, PSC, chronic pouchitis, CD of the pouch, chronic cuffitis, and family history of CRC).*

**Synopsis.** Proposed risk factors for pouch neoplasia include PSC, chronic pouchitis, CD of the pouch, chronic cuffitis, and family history of CRC. None of these has been confirmed in studies with statistical confidence because of limitations in sample size.[106,107] The International Ileal Pouch Consortium recommended surveillance pouchoscopy every 1 to 3 years for these patients.[84]

**Statement 27.** *Surveillance pouchoscopy should be performed every 3 years in IBD patients at average risk for dysplasia.*

**Synopsis.** The frequency of routine surveillance pouchoscopy in asymptomatic patients at average risk is controversial. Some authors do not recommend surveillance pouchoscopy in asymptomatic patients because of the rarity of dysplasia.[121] The arguments for surveillance pouchoscopy for these patients are the poorly defined natural history of pouch neoplasia, dismal prognosis of pouch cancer, and additional diagnostic and therapeutic benefits

in finding other benign pouch disorders during surveillance pouchoscopy.[122] Therefore, the International Ileal Pouch Consortium recommends surveillance pouchoscopy every 3 years for this group of patients.[84]

**Statement 28.** *High-definition (HD) white-light (WL) endoscopy or image-enhanced endoscopy (such as virtual or dye-based chromoendoscopy) with targeted or random biopsy sampling should be used for surveillance colonoscopy*.

**Synopsis.** Since the publication of the Surveillance for Colorectal Endoscopic Neoplasia Detection and Management in IBD patients consensus conference in 2015,[78] multiple articles have been published comparing outcomes of chromoendoscopy and WL colonoscopy in the surveillance of CAN.[123-126] A network meta-analysis of 6 prospective randomized controlled trials of 1038 patients showed the superiority of narrow-band imaging with HD over WL imaging with HD and chromoendoscopy with HD over standard-definition WL endoscopy on direct meta-analysis. The rank order of the best modality was HD narrow-band imaging, chromoendoscopy, and WL endoscopy and standard-definition WL endoscopy on network meta-analysis.[127] A separate meta-analysis of 7 randomized controlled trials of 2457 patients showed the superiority of dye-based chromoendoscopy to standard-definition WL endoscopy. However, no differences in the number of patients diagnosed with $\geq$1 dysplastic lesions, total number of dysplastic lesions detected, and number of dysplastic lesions detected by directed biopsy sampling between dye-based chromoendoscopy and virtual chromoendoscopy were reported.[128] Advantages of imaging-enhanced targeted endoscopy include a reduction in the number of biopsy samples, shorter procedure time, and lower rate of biopsy sampling–associated adverse events.

**Statement 29.** *HD WL endoscopy or image-enhanced endoscopy (such as virtual or dye-based chromoendoscopy) with targeted and random biopsy sampling should be used for surveillance colonoscopy in high-risk patients, such as those with PSC.*

**Synopsis.** Despite advances in imaging technology, some dysplastic lesions may be endoscopically invisible (ie, dysplasia diagnosed histologically from random biopsy sampling without an associated discrete lesion), especially in those patients with nonconventional dysplasia variants such as hypermucinous, goblet cell–deficient, and crypt cell dysplasia, such that targeted biopsy sampling alone may put the patients at risk (Fig. 5).[129] In a study comparing IBD patients with and without PSC, those with PSC were more likely to develop dysplasia, and when dysplasia occurred, it often had nonconventional dysplastic features with invisible endoscopic gross appearance.[130] In addition to PSC, patients with tubular colon and rectum or a history of CAN (particularly invisible dysplasia) may also benefit from HD WL endoscopy or image-enhanced endoscopy with both targeted and random biopsy sampling.

## INTERVENTIONAL IBD

Endoscopic therapy or interventional IBD plays a growing role in the management of complex IBD. The main indications for interventional IBD are strictures; fistulas and abscesses; intraluminal lesions, bezoars, and foreign bodies; IBD surgery–associated adverse events; and CAN.[131,132] Attempts have been made to standardize the practice of interventional IBD.[47,133] Interventional therapies are done by select endoscopists at select centers that have additional specialized IBD surgeons.

### Endoscopic therapy of IBD strictures

**Statement 30.** *Intralesional injection of long-acting steroids is not recommended before, during, or after endoscopic balloon dilation (EBD) for strictures.*

**Synopsis.** EBD has become a standard of care in the treatment of primary or anastomotic strictures in IBD.[134,135] Conflicting results have been reported regarding intralesional injection of steroids at endoscopic stricture dilations in CD from 2 small randomized controlled trials[136,137] and other case series. A panel of experts did not recommend intralesional injection of long-acting corticosteroids after EBD in Crohn's strictures.[121]

**Statement 31.** *EBD of deeply ulcerated strictures should be avoided.*

**Synopsis.** Although the association between EBD of deeply ulcerated strictures and efficacy and risk of bleeding or perforation has not been confirmed, endoscopic therapy in this type of stricture should be avoided.[121,123]

**Statement 32.** *The long-term success of endoscopic stricture therapy is measured by reintervention-free survival and surgery-free survival.*

**Synopsis.** Reinterventions (ie, endoscopic retreatment–free survival and surgery-free survival) have been frequently used as measures of efficacy for endoscopic therapy for strictures.[138-140] Improvement in symptoms and symptom-free survival has also been used as short- and long- term outcome measurements.[141-143] When reporting outcomes, the endoscopist should also describe procedure-associated adverse events, mainly bleeding, perforation, and ileus or aspiration.

Whether endoscopic stricture treatment should be performed in asymptomatic patients with IBD-associated strictures is controversial. The rationale for endoscopic treatment of strictures in asymptomatic patients is that the patient's clinical symptoms are often not correlated with objective findings, and asymptomatic strictures, if left alone, can lead to prestenotic luminal dilation, acute intestinal obstruction, or even fistula formation.[121] Subtle progression of CD strictures may eventually lead to surgical resection with loss of opportunity for early endoscopic intervention. For the management of CD stricture, EBD was shown to be more cost-effective than surgical resection and anastomosis.[144]

**Statement 33.** *Large (>1 cm) inflammatory polyps may be removed to reduce the symptoms of bleeding,*

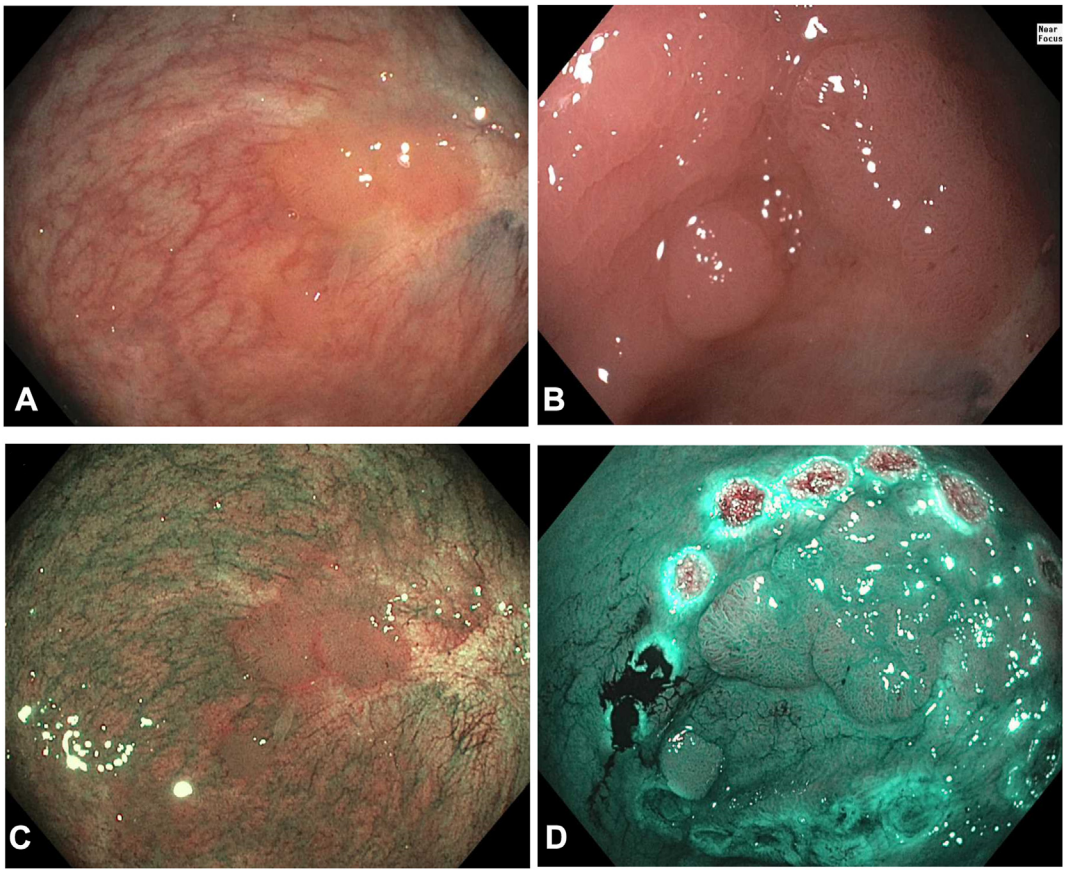


**Figure 5.** Image-enhanced endoscopy in the detection of colitis-associated neoplasia (here it is a multifocal low-grade dysplastic lesion in the rectum). **A,** White light. **B,** Magnified endoscopy. **C,** Narrow-band imaging (NBI). **D,** Dye-based and virtual (NBI) chromoendoscopy helped to mark the lesion for endoscopic submucosal dissection.

*obstruction, and anemia, even though the risk of dysplasia is low.*

**Synopsis.** A meta-analysis of 164 studies for CRC in IBD showed that the presence of postinflammatory polyps is a risk factor for the development of neoplasia with an odds ratio of 2.54 (95% CI, 1.40-4.60).[110] Inflammatory polyps in IBD can be a contributing factor to anemia[145,146] or obstruction.[147]

**Statement 34.** *Endoscopic stents should not be used in the treatment of benign disease-associated or anastomotic strictures in pre- or postoperative CD or UC.*

**Synopsis.** A recent randomized controlled trial of self-expandable metal stents failed to show more favorable efficacy and safety than EBD in treating CD strictures.[148]

**Statement 35.** *Surgical intervention should occur for CD strictures, especially long (>4-5 cm) or complex (eg, fistula and/or abscess-associated strictures, those refractory to previous intervention, or recurrent [requiring endoscopic intervention more often than every 3-6 months]) strictures after previously successful endoscopic interventions.*

**Synopsis.** The decision on endoscopic versus surgical therapy is determined by patient (eg, comorbidities), disease (CD vs UC, concurrent inflammation, and medications), stricture (eg, primary vs secondary, short or long [with a cutoff 4-5 cm], simple vs complex), endoscopist, and surgical (eg, setting and local expertise) factors. Expert consensus has recommended long (>4-5 cm) or complex IBD strictures may benefit more from surgical intervention than endoscopic therapy.[121,123]

## Endoscopic treatment of CAN

**Statement 36.** *Polypectomy, EMR, or endoscopic submucosal dissection may be performed on polypoid or raised, liftable dysplastic lesions with a clear, well-defined border.*

**Synopsis.** A meta-analysis of 7 studies in 506 patients and 610 lesions showed a pooled rate of complete endoscopic resection of 97.9% (95% CI, 95.3-99.7). The pooled rate of endoscopic perforation was .8% (95% CI, .1-2.2), whereas bleeding occurred in 1.6% of patients (95% CI, .4-3.3). Overall, 6.6% of patients (95% CI, 3.6-10.2) underwent surgery after endoscopic resection.[149] The main criteria for EMR or endoscopic submucosal dissection in CAN are lesions that are polypoid or raised, liftable with submucosal injection of injectable substance, dysplastic, and with a clear well-defined border. A biopsy sample of the base of the polypoid or resectable lesion should be

performed. In addition, biopsy sampling of the margin should be performed if the lesion is removed by piecemeal polypectomy. Care should be taken to ensure the EMR and endoscopic submucosal dissection specimens are appropriately processed for histology.

## FUTURE PERSPECTIVES

**Statement 37.** *The exploration of artificial intelligence in the diagnosis and surveillance of IBD should continue.*

**Synopsis.** Artificial intelligence–assisted endoscopy (including colonoscopy and capsule endoscopy) and histology have been investigated for diagnosis,[150,151] differential diagnosis,[152] disease monitoring, and prognostication.[153-156] Computer-assisted imaging may enhance the quantification of global endoscopic inflammation scores.[157] The emerging use of artificial intelligence in IBD will include surveillance of CAN.

**Statement 38.** *Applications of novel endoscopic devices in interventional IBD (eg, drug-coated balloons, scope-tip devices, clipping or suturing devices, and endoscopic delivery systems for cell therapy) should be explored.*

**Synopsis.** A major challenge in interventional IBD is the management of refractory primary or anastomotic strictures. In addition to endoscopic dilation with a mechanical balloon and electroincision, drug-coated balloon dilation of CD strictures[158] and endoscopic injection of mesenchymal stem cells[159] have been investigated. Clipping or suture devices have been used for the anastomotic leaks in CD[160] or UC.[161]

## SUMMARY AND CONCLUSIONS

GI endoscopy plays a key role in the diagnosis, differential diagnosis, assessment of disease activity and treatment response, dysplasia surveillance, and delivery of targeted therapy. The field is evolving rapidly. By better understanding the etiopathogenesis and disease course and advances in medical, endoscopic, and surgical therapy and advanced endoscopic technology, we will be able to recognize and provide direction on currently controversial statements.

## DISCLOSURE

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## REFERENCES

1. Ng SC, Shi HY, Hamidi N, et al. Worldwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies. Lancet 2017;390:2769-78.

2. Okoli C, Pawlowski SD. The Delphi method as a research tool: an example, design considerations and applications. Inf Manag 2004;42:15-29.
3. Aceves SS, Alexander JA, Baron TH, et al. Endoscopic approach to eosinophilic esophagitis: American Society for Gastrointestinal Endoscopy consensus conference. Gastrointest Endosc 2022;96:576-92.
4. Silverberg MS, Satsangi J, Ahmad T, et al. Toward an integrated clinical, molecular and serological classification of inflammatory bowel disease: report of a Working Party of the 2005 Montreal World Congress of Gastroenterology. Can J Gastroenterol 2005;19(Suppl A):5A-36A.
5. Jeong SH, Lee KJ, Kim YB, et al. Diagnostic value of terminal ileum intubation during colonoscopy. J Gastroenterol Hepatol 2008;23:51-5.
6. Leighton JA, Shen B, Baron TH, et al. ASGE guideline: endoscopy in the diagnosis and treatment of inflammatory bowel disease. Gastrointest Endosc 2006;63:558-65.
7. Makkar R, Lopez R, Shen B. Clinical utility of retrograde terminal ileum intubation in the evaluation of chronic non-bloody diarrhea. J Dig Dis 2013;14:536-42.
8. Bruining DH, Oliva S, Fleisher MR, Fischer M, Fletcher JG; BLINK study group. Panenteric capsule endoscopy versus ileocolonoscopy plus magnetic resonance enterography in Crohn's disease: a multicentre, prospective study. BMJ Open Gastroenterol 2020;7:e000365.
9. Turner K, Genta RM, Lujan G, et al. Significance of the epithelioid granuloma in biopsies of Crohn's colitis. Inflamm Bowel Dis 2014;20:2271-5.
10. Rubio CA, Orrego A, Nesi G, et al. Frequency of epithelioid granulomas in colonoscopic biopsy specimens from paediatric and adult patients with Crohn's colitis. J Clin Pathol 2007;60:1268-72.
11. Nguyen J, Lee S, Yang GY. Comprehensive evaluation and unique morphologic features of nonsteroidal anti-inflammatory drug (NSAID) enteropathy in the terminal ileum. Int J Surg Pathol 2022;30:616-22.
12. Matsumoto T, Nakamura S, Esaki M, et al. Endoscopic features of chronic nonspecific multiple ulcers of the small intestine: comparison with nonsteroidal anti-inflammatory drug-induced enteropathy. Dig Dis Sci 2006;51:1357-63.
13. ASGE Standards of Practice Committee; Saltzman JR, Cash BD, Pasha SF, et al. Bowel preparation before colonoscopy. Gastrointest Endosc 2015;81:781-94.
14. Wexner SD, Beck DE, Baron TH, et al. A consensus document on bowel preparation before colonoscopy: prepared by a task force from the American Society of Colon and Rectal Surgeons (ASCRS), the American Society for Gastrointestinal Endoscopy (ASGE), and the Society of American Gastrointestinal and Endoscopic Surgeons (SAGES). Gastrointest Endosc 2006;63:894-909.
15. Rejchrt S, Bures J, Siroký M, et al. A prospective, observational study of colonic mucosal abnormalities associated with orally administered sodium phosphate for colon cleansing before colonoscopy. Gastrointest Endosc 2004;59:651-4.
16. Bernstein CN, Shanahan F, Anton PA, et al. Patchiness of mucosal inflammation in treated ulcerative colitis: a prospective study. Gastrointest Endosc 1995;42:232-7.
17. Cleynen I, Boucher G, Jostins L, et al. Inherited determinants of Crohn's disease and ulcerative colitis phenotypes: a genetic association study. Lancet 2016;387:156-67.
18. Tanaka M, Saito H, Kusumi T, et al. Spatial distribution and histogenesis of colorectal Paneth cell metaplasia in idiopathic inflammatory bowel disease. J Gastroenterol Hepatol 2001;16:1353-9.
19. Tokuyama M, Dhingra S, Polydorides AD. Clinicopathologic features and diagnostic implications of pyloric gland metaplasia in intestinal specimens. Am J Surg Pathol 2021;45:365-73.
20. Schembri J, Bonello J, Christodoulou DK, et al. Segmental colitis associated with diverticulosis: Is it the coexistence of colonic diverticulosis and inflammatory bowel disease? Ann Gastroenterol 2017;30:257-61.
21. Reggiani Bonetti L, Leoncini G, Daperno M, et al. Histopathology of non-IBD colitis practical recommendations from pathologists of IG-IBD Group. Dig Liver Dis 2021;53:950-7.
22. Geboes K, Riddell R, Ost A, et al. A reproducible grading scale for histological assessment of inflammation in ulcerative colitis. Gut 2000;47:404-9.
23. Marchal-Bressenot A, Salleron J, Boulagnon-Rombi C, et al. Development and validation of the Nancy Histological Index for UC. Gut 2017;66:43-9.
24. Mosli MH, Feagan BG, Zou G, et al. Development and validation of a histological index for UC. Gut 2017;66:50-8.
25. McBride RB, Suarez-Farinas M, Ko HM, et al. Density of biopsy sampling required to ensure accurate histological assessment of inflammation in active ulcerative colitis. Inflamm Bowel Dis 2023;29:1706-12.
26. Xu C, Jiang W, Wang L, et al. Intestinal ultrasound for differentiating fibrotic or inflammatory stenosis in Crohn's disease: a systematic review and meta-analysis. J Crohns Colitis 2022;16:1493-504.
27. Kellar A, Dolinger M, Novak KL, et al. Intestinal ultrasound for the pediatric gastroenterologist: a guide for inflammatory bowel disease monitoring in children: expert consensus on behalf of the International Bowel Ultrasound Group (IBUS) Pediatric Committee. J Pediatr Gastroenterol Nutr 2023;76:142-8.
28. Ilvemark JFKF, Hansen T, Goodsall TM, et al. Defining transabdominal intestinal ultrasound treatment response and remission in inflammatory bowel disease: systematic review and expert consensus statement. J Crohns Colitis 2022;16:554-80.
29. Kucharzik T, Wilkens R, D'Agostino MA, et al. Early ultrasound response and progressive transmural remission after treatment with ustekinumab in Crohn's disease. Clin Gastroenterol Hepatol 2023;21:153-63.
30. Prentice R, Wright EK, Flanagan E, et al. The use of fecal calprotectin and intestinal ultrasound in the evaluation and management of stricturing Crohn's disease in pregnancy. Inflamm Bowel Dis 2022;28:e13-6.
31. Kovacs M, Muller KE, Arato A, et al. Diagnostic yield of upper endoscopy in paediatric patients with Crohn's disease and ulcerative colitis. Subanalysis of the HUPIR registry. J Crohns Colitis 2012;6:86-94.
32. Niv Y. Small-bowel mucosal healing assessment by capsule endoscopy as a predictor of long-term clinical remission in patients with Crohn's disease: a systematic review and meta-analysis. Eur J Gastroenterol Hepatol 2017;29:844-8.
33. Pasha SF, Pennazio M, Rondonotti E, et al. Capsule retention in Crohn's disease: a meta-analysis. Inflamm Bowel Dis 2020;26:33-42.
34. Turner D, Ricciuto A, Lewis A, et al. STRIDE-II: an update on the Selecting Therapeutic Targets in Inflammatory Bowel Disease (STRIDE) initiative of the International Organization for the Study of IBD (IOIBD): determining therapeutic goals for treat-to-target strategies in IBD. Gastroenterology 2021;160:1570-83.
35. Mary JY, Modigliani R. Development and validation of an endoscopic index of the severity for Crohn's disease: a prospective multicentre study. Groupe d'Etudes Therapeutiques des Affections Inflammatoires du Tube Digestif (GETAID). Gut 1989;30:983-9.
36. Daperno M, D'Haens G, Van Assche G, et al. Development and validation of a new, simplified endoscopic activity score for Crohn's disease: the SES-CD. Gastrointest Endosc 2004;60:505-12.
37. Truelove SC, Witts LJ. Cortisone in ulcerative colitis; final report on a therapeutic trial. Br Med J 1955;2:1041-8.
38. Schroeder KW, Tremaine WJ, Ilstrup DM. Coated oral 5-aminosalicylic acid therapy for mildly to moderately active ulcerative colitis. A randomized study. N Engl J Med 1987;317:1625-9.
39. Rutgeerts P, Geboes K, Vantrappen G, et al. Predictability of the postoperative course of Crohn's disease. Gastroenterology 1990;99:956-63.
40. Chongthammakun V, Fialho A, Fialho A, et al. Correlation of the Rutgeerts score and recurrence of Crohn's disease in patients with end ileostomy. Gastroenterol Rep 2017;5:271-6.
41. Shen B, Plesec TP, Remer E, et al. Asymmetric endoscopic inflammation of the ileal pouch: a sign of ischemic pouchitis? Inflamm Bowel Dis 2010;16:836-46.

42. Parsi MA, Shen B, Achkar JP, et al. Fecal lactoferrin for diagnosis of symptomatic patients with ileal pouch-anal anastomosis. Gastroenterology 2004;126:1280-6.
43. McKechnie T, Lee Y, Kruse C, et al. The role of fecal calprotectin in the diagnosis of acute pouchitis following IPAA for ulcerative colitis: a systematic clinical review. Int J Colorectal Dis 2020;35:1619-28.
44. Yamamoto T, Shimoyama T, Bamba T, et al. Consecutive monitoring of fecal calprotectin and lactoferrin for the early diagnosis and prediction of pouchitis after restorative proctocolectomy for ulcerative colitis. Am J Gastroenterol 2015;110:881-7.
45. Ollech JE, Bannon L, Maharshak N, et al. Fecal calprotectin is increased in pouchitis and progressively increases with more severe endoscopic and histologic disease. Clin Gastroenterol Hepatol 2022;20:1839-46.
46. Cohen NA, Steinberg JM, Silfen A, et al. Endo-histologic normalization is achievable with tofacitinib and is associated with improved clinical outcomes. Dig Dis Sci 2023;68:1464-72.
47. CORE-IBD Collaborators; Ma C, Hanzel J, et al. CORE-IBD: a multidisciplinary international consensus initiative to develop a core outcome set for randomized controlled trials in inflammatory bowel disease. Gastroenterology 2022;163:950-64.
48. Weinstein-Nakar I, Focht G, Church P, et al; ImageKids Study Group. Associations among mucosal and transmural healing and fecal level of calprotectin in children with Crohn's disease. Clin Gastroenterol Hepatol 2018;16:1089-97.
49. Bertani L, Blandizzi C, Mumolo MG, et al. Fecal calprotectin predicts mucosal healing in patients with ulcerative colitis treated with biological therapies: a prospective study. Clin Transl Gastroenterol 2020;11:e00174.
50. Mourad FH, Hashash JG, Kariyawasam VC, et al. Ulcerative colitis and cytomegalovirus infection: from A to Z. J Crohns Colitis 2020;14:1162-71.
51. Kucharzik T, Ellul P, Greuter T, et al. ECCO guidelines on the prevention, diagnosis, and management of infections in inflammatory bowel disease. J Crohns Colitis 2021;15:879-91.
52. McCurdy JD, Enders FT, Jones A, et al. Detection of cytomegalovirus in patients with inflammatory bowel disease: where to biopsy and how many biopsies? Inflamm Bowel Dis 2015;21:2833-8.
53. Clos-Parals A, Rodríguez-Martínez P, Cañete F, et al. Prognostic value of the burden of cytomegalovirus colonic reactivation evaluated by immunohistochemical staining in patients with active ulcerative colitis. J Crohns Colitis 2019;13:385-8.
54. Zidar N, Ferkolj I, Tepeš K, et al. Diagnosing cytomegalovirus in patients with inflammatory bowel disease—by immunohistochemistry or polymerase chain reaction? Virchows Arch 2015;466:533-9.
55. Shen B, Kochhar GS, Navaneethan U, et al. Endoscopic evaluation of surgically altered bowel in inflammatory bowel disease: a consensus guideline from the Global Interventional Inflammatory Bowel Disease Group. Lancet Gastroenterol Hepatol 2021;6:482-97. [Erratum: Lancet Gastroenterol Hepatol 2021 Jun6:e5.].
56. Akiyama S, Yamada A, Ollech JE, et al. Predictability of simple endoscopic score for Crohn's disease for postoperative outcomes in Crohn's disease. J Gastroenterol Hepatol 2021;36:2785-93.
57. De Cruz P, Kamm MA, Hamilton AL, et al. Crohn's disease management after intestinal resection: a randomised trial. Lancet 2015;385:1406-17.
58. Shen B, Kochhar GS, Kariv R, et al. Diagnosis and classification of ileal pouch disorders: consensus guidelines from the International Ileal Pouch Consortium. Lancet Gastroenterol Hepatol 2021;6:826-49.
59. Barnes EL, Agrawal M, Syal G, et al. AGA clinical practice guideline on the management of pouchitis and inflammatory pouch disorders. Gastroenterology 2024;166:59-85.
60. Shen B, Achkar JP, Lashner BA, et al. Endoscopic and histologic evaluation together with symptom assessment are required to diagnose pouchitis. Gastroenterology 2001;121:261-7.
61. Ben-Bassat O, Tyler AD, Xu W, et al. Ileal pouch symptoms do not correlate with inflammation of the pouch. Clin Gastroenterol Hepatol 2014;12:831-7.
62. Akiyama S, Ollech JE, Rai V, et al. Endoscopic phenotype of the J pouch in patients with inflammatory bowel disease: a new classification for pouch outcomes. Clin Gastroenterol Hepatol 2022;20:293-302.
63. Sandborn WJ, Tremaine WJ, Batts KP, et al. Pouchitis after ileal pouch-anal anastomosis: a Pouchitis Disease Activity Index. Mayo Clin Proc 1994;69:409-15.
64. Shen B, Fazio VW, Remzi FH, et al. Effect of withdrawal of nonsteroidal anti-inflammatory drug use on ileal pouch disorders. Dig Dis Sci 2007;52:3321-8.
65. Shen B, Lashner BA, Bennett AE, et al. Treatment of rectal cuff inflammation (cuffitis) in patients with ulcerative colitis following restorative proctocolectomy and ileal pouch-anal anastomosis. Am J Gastroenterol 2004;99:1527-31.
66. Samaan MA, Shen B, Mosli MH, et al. Reliability among central readers in the evaluation of endoscopic disease activity in pouchitis. Gastrointest Endosc 2018;88:360-9.
67. Wang Z, Wang J, Yang Z, Li S, Ding C, Gong J. A specific phenotype of pouchitis was associated with worst prognosis in patients with ulcerative colitis according to Chicago classification. Dig Liver Dis 2024;56:1007-13.
68. McCurdy JD, Loftus EV Jr, Tremaine WJ, et al. Cytomegalovirus infection of the ileoanal pouch: clinical characteristics and outcomes. Inflamm Bowel Dis 2013;19:2394-9.
69. Del Valle JP, Lee GC, Serrato JC, et al. Recurrence of *Clostridium difficile* and cytomegalovirus infections in patients with ulcerative colitis who undergo ileal pouch-anal anastomosis. Dig Dis Sci 2021;66:4441-7.
70. Shen B, Ko HM, Ma H, Kiran R, Church J. Solitary pouch ulcer syndrome-a newly recognized phenotype of the ileal pouch disorders. Gastroenterol Rep (Oxf) 2024;12:goae073.
71. Liu G, Ma J, Liu X, et al. Clinical implications of noncaseating granulomas on histology in patients with ileal pouches. Inflamm Bowel Dis 2015;21:1801-8.
72. Shen B, Achkar JP, Lashner BA, et al. Endoscopic and histologic evaluation together with symptom assessment are required to diagnose pouchitis. Gastroenterology 2001;121:261-7.
73. Shen B, Achkar JP, Connor JT, et al. Modified pouchitis disease activity index: a simplified approach to the diagnosis of pouchitis. Dis Colon Rectum 2003;46:748-53.
74. Travis S, Silverberg MS, Danese S, et al. EARNEST Study Group. Vedolizumab for the treatment of chronic pouchitis. N Engl J Med 2023;388:1191-200.
75. Daoud ND, Hashash JG, Picco MF, et al. Faecal calprotectin from ileostomy output is sensitive and specific for the prediction of small bowel inflammation in patients with Crohn's disease. J Crohns Colitis 2022;16:601-5.
76. Shaffer SR, Erondu AI, Traboulsi C, et al. Achieving histologic normalization in ulcerative colitis is associated with a reduced risk of subsequent dysplasia. Inflamm Bowel Dis 2022;28:553-9.
77. Colman RJ, Rubin DT. Histological inflammation increases the risk of colorectal neoplasia in ulcerative colitis: a systematic review. Intest Res 2016;14:202-10.
78. Yvellez OV, Rai V, Sossenheimer PH, et al. Cumulative histologic inflammation predicts colorectal neoplasia in ulcerative colitis: a validation study. Inflamm Bowel Dis 2021;27:203-6.
79. Shah SC, Itzkowitz SH. Colorectal cancer in inflammatory bowel disease: mechanisms and management. Gastroenterology 2022;162:715-30.
80. Navaneethan U, Jegadeesan R, Gutierrez NG, et al. Progression of low-grade dysplasia to advanced neoplasia based on the location and morphology of dysplasia in ulcerative colitis patients with extensive colitis under colonoscopic surveillance. J Crohns Colitis 2013;7:e684-91.

81. Fumery M, Dulai PS, Gupta S, et al. Incidence, risk factors, and outcomes of colorectal cancer in patients with ulcerative colitis with low-grade dysplasia: a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2017;15:665-74.
82. Loftus EV Jr, Harewood GC, Loftus CG, et al. PSC-IBD: a unique form of inflammatory bowel disease associated with primary sclerosing cholangitis. Gut 2005;54:91-6.
83. Loftus EV Jr, Sandborn WJ, Tremaine WJ, et al. Risk of colorectal neoplasia in patients with primary sclerosing cholangitis. Gastroenterology 1996;110:432-40.
84. Aune D, Sen A, Norat T, et al. Primary sclerosing cholangitis and the risk of cancer, cardiovascular disease, and all-cause mortality: a systematic review and meta-analysis of cohort studies. Sci Rep 2021;11:10646.
85. Farraye FA, Odze RD, Eaden J, et al. AGA medical position statement on the diagnosis and management of colorectal neoplasia in inflammatory bowel disease. Gastroenterology 2010;138:738-45.
86. Laine L, Kaltenbach T, Barkun A, et al. SCENIC international consensus statement on surveillance and management of dysplasia in inflammatory bowel disease. Gastroenterology 2015;148:639-51.
87. Lamb CA, Kennedy NA, Raine T, et al. British Society of Gastroenterology consensus guidelines on the management of inflammatory bowel disease in adults. Gut 2019;68(Suppl 3):s1-106. [Erratum: Gut 2021 Apr70:1.].
88. Murthy SK, Feuerstein JD, Nguyen GC, et al. AGA Clinical practice update on endoscopic surveillance and management of colorectal dysplasia in inflammatory bowel diseases: expert review. Gastroenterology 2021;161:1043-51.
89. Wang AY, Hwang JH, Bhatt A, et al. AGA clinical practice update on surveillance after pathologically curative endoscopic submucosal dissection of early gastrointestinal neoplasia in the United States: commentary. Gastroenterology 2021;161:2030-40.
90. Ward D, Neumann A, Hendel JW, et al. Danish Society for Gastroenterology and Hepatology's clinical recommendations for colonoscopic surveillance for colorectal dysplasia and cancer in patients with inflammatory bowel disease. Scand J Gastroenterol 2022;57:457-64.
91. Rubin DT, Ananthakrishnan AN, Siegel CA, et al. ACG clinical guideline: ulcerative colitis in adults. Am J Gastroenterol 2019;114:384-413.
92. Kiran RP, Kochhar GS, Kariv R, et al. Management of pouch neoplasia: consensus guidelines from the International Ileal Pouch Consortium. Lancet Gastroenterol Hepatol 2022;7:871-93.
93. Adamina M, Feakins R, Iacucci M, et al. ECCO topical review optimising reporting in surgery, endoscopy, and histopathology. J Crohns Colitis 2021;15:1089-105.
94. Harpaz N, Itzkowitz SH. Pathology and clinical significance of inflammatory bowel disease-associated colorectal dysplastic lesions. Gastroenterol Clin North Am 2024;53:133-54.
95. Magro F, Gionchetti P, Eliakim R, et al. European Crohn's and Colitis Organisation [ECCO]. Third European evidence-based consensus on diagnosis and management of ulcerative colitis. Part 1: definitions, diagnosis, extra-intestinal manifestations, pregnancy, cancer surveillance, surgery, and ileo-anal pouch disorders. J Crohns Colitis 2017;11:649-70.
96. Lamb CA, Kennedy NA, Raine T, et al. British Society of Gastroenterology consensus guidelines on the management of inflammatory bowel disease in adults. Gut 2019;68(Suppl 3):s1-106.
97. Leoncini G, Donato F, Reggiani-Bonetti L, et al; IG-IBD Pathology Group. Diagnostic interobserver variability in Crohn's disease- and ulcerative colitis-associated dysplasia: a multicenter digital survey from the IG-IBD Pathologists Group. Tech Coloproctol 2021;25:101-8.
98. Friedberg S, Rubin DT. Intestinal cancer and dysplasia in Crohn's disease. Gastroenterol Clin North Am 2022;51:369-79.
99. Gatenby G, Glyn T, Pearson J, et al. The long-term incidence of dysplasia and colorectal cancer in a Crohn's colitis population-based cohort. Colorectal Dis 2021;23:2399-406.
100. Kiran RP, Nisar PJ, Goldblum JR, et al. Dysplasia associated with Crohn's colitis: segmental colectomy or more extended resection? Ann Surg 2012;256:221-6.
101. Maykel JA, Hagerman G, Mellgren AF, et al. Crohn's colitis: the incidence of dysplasia and adenocarcinoma in surgical patients. Dis Colon Rectum 2006;49:950-7.
102. Maser EA, Sachar DB, Kruse D, et al. High rates of metachronous colon cancer or dysplasia after segmental resection or subtotal colectomy in Crohn's colitis. Inflamm Bowel Dis 2013;19:1827-32.
103. Rubin CE, Haggitt RC, Burmer GC, et al. DNA aneuploidy in colonic biopsies predicts future development of dysplasia in ulcerative colitis. Gastroenterology 1992;103:1611-20.
104. Iannone A, Ruospo M, Wong G, et al. Chromoendoscopy for surveillance in ulcerative colitis and Crohn's disease: a systematic review of randomized trials. Clin Gastroenterol Hepatol 2017;15:1684-97.
105. Mooiweer E, van der Meulen-de Jong AE, Ponsioen CY, et al. Chromoendoscopy for surveillance in inflammatory bowel disease does not increase neoplasia detection compared with conventional colonoscopy with random biopsies: results from a large retrospective study. Am J Gastroenterol 2015;110:1014-21.
106. Azizi S, Al-Rubaye H, Turki MAA, et al. Detecting dysplasia using white light endoscopy or chromoendoscopy in ulcerative colitis patients without primary sclerosing cholangitis: a systematic review and meta-analysis. Int J Surg 2018;52:180-8.
107. Gordon IO, Agrawal N, Goldblum JR, et al. Fibrosis in ulcerative colitis: mechanisms, features, and consequences of a neglected problem. Inflamm Bowel Dis 2014;20:2198-206.
108. Xu W, Ding W, Gu Y, et al. Risk factors of colorectal stricture associated with developing high-grade dysplasia or cancer in ulcerative colitis: a multicenter long-term follow-up study. Gut Liver 2020;14:601-10.
109. Zheng HH, Jiang XL. Increased risk of colorectal neoplasia in patients with primary sclerosing cholangitis and inflammatory bowel disease: a meta-analysis of 16 observational studies. Eur J Gastroenterol Hepatol 2016;28:383-90.
110. Wijnands AM, de Jong ME, Lutgens MWMD, et al. Prognostic factors for advanced colorectal neoplasia in inflammatory bowel disease: systematic review and meta-analysis. Gastroenterology 2021;160:1584-98.
111. Moussata D, Allez M, Cazals-Hatem D, et al. Are random biopsies still useful for the detection of neoplasia in patients with IBD undergoing surveillance colonoscopy with chromoendoscopy? Gut 2018;67:616-24.
112. Sicilia B, Vicente R, Arias L, et al. Recommendations of the Spanish Working Group on Crohn's disease and Ulcerative Colitis (Grupo Español de Trabajo en Enfermedad de Crohn y Colitis Ulcerosa - GETECCU) on dysplasia screening in inflammatory bowel disease patients. Gastroenterol Hepatol 2021;44:435-47.
113. Kiran RP, Khoury W, Church JM, et al. Colorectal cancer complicating inflammatory bowel disease: similarities and differences between Crohn's and ulcerative colitis based on three decades of experience. Ann Surg 2010;252:330-5.
114. Elmahdi R, Lemser CE, Thomsen SB, et al. Development of cancer among patients with pediatric-onset inflammatory bowel disease: a meta-analysis of population-based studies. JAMA Netw Open 2022;5:e220595.
115. Bettner W, Rizzo A, Brant S, et al. Low incidence of dysplasia and colorectal cancer observed among inflammatory bowel disease patients with prolonged colonic diversion. Inflamm Bowel Dis 2018;24:1092-8.
116. Dal Buono A, Carvello M, Sachar DB, et al. Diversion proctocolitis and the problem of the forgotten rectum in inflammatory bowel diseases: a systematic review. United Eur Gastroenterol J 2021;9:1157-67.
117. Shen B, Kochhar GS, Navaneethan U, et al. Endoscopic evaluation of surgically altered bowel in inflammatory bowel disease: a consensus guideline from the Global Interventional Inflammatory Bowel Disease Group. Lancet Gastroenterol Hepatol 2021;6:482-97.

118. Kariv R, Remzi FH, Lian L, et al. Preoperative colorectal neoplasia increases risk for pouch neoplasia in patients with restorative proctocolectomy. Gastroenterology 2010;139:806-12.
119. Derikx LA, Kievit W, Drenth JP, et al. Prior colorectal neoplasia is associated with increased risk of ileoanal pouch neoplasia in patients with inflammatory bowel disease. Gastroenterology 2014;146:119-28.
120. Wu XR, Remzi FH, Liu XL, et al. Disease course and management strategy of pouch neoplasia in patients with underlying inflammatory bowel diseases. Inflamm Bowel Dis 2014;20:2073-82.
121. Lightner AL, Vaidya P, Vogler S, et al. Surveillance pouchoscopy for dysplasia: Cleveland Clinic Ileoanal Pouch Anastomosis database. Br J Surg 2020;107:1826-31.
122. Zhu H, Wu XR, Queener E, et al. Clinical value of surveillance pouchoscopy in asymptomatic ileal pouch patients with underlying inflammatory bowel disease. Surg Endosc 2013;27:4325-32.
123. Bisschops R, Bessissow T, Dekker E, et al. Pit pattern analysis with high-definition chromoendoscopy and narrow-band imaging for optical diagnosis of dysplasia in patients with ulcerative colitis. Gastrointest Endosc 2017;86:1100-6.
124. Bisschops R, Bessissow T, Joseph JA, et al. Chromoendoscopy versus narrow band imaging in UC: a prospective randomised controlled trial. Gut 2018;67:1087-94.
125. Vleugels JLA, Rutter MD, Ragunath K, et al. Diagnostic accuracy of endoscopic trimodal imaging and chromoendoscopy for lesion characterization in ulcerative colitis. J Crohns Colitis 2018;12:1438-47.
126. Har-Noy O, Katz L, Avni T, et al. Chromoendoscopy, narrow-band imaging or white light endoscopy for neoplasia detection in inflammatory bowel diseases. Dig Dis Sci 2017;62:2982-90.
127. Gondal B, Haider H, Komaki Y, et al. Efficacy of various endoscopic modalities in detecting dysplasia in ulcerative colitis: a systematic review and network meta-analysis. World J Gastrointest Endosc 2020;12:159-71.
128. Resende RH, Ribeiro IB, de Moura DTH, et al. Surveillance in inflammatory bowel disease: Is chromoendoscopy the only way to go? A systematic review and meta-analysis of randomized clinical trials. Endosc Int Open 2020;8:E578-90.
129. Choi WT, Salomao M, Zhao L, et al. Hypermucinous, goblet cell-deficient and crypt cell dysplasias in inflammatory bowel disease are often associated with flat/invisible endoscopic appearance and advanced neoplasia on follow-up. J Crohns Colitis 2022;16:98-108.
130. Zhang R, Lauwers GY, Choi WT. Increased risk of non-conventional and invisible dysplasias in patients with primary sclerosing cholangitis and inflammatory bowel disease. J Crohns Colitis 2022;1:1825-34.
131. Shen B, Kochhar G, Navaneethan U, et al. Role of interventional inflammatory bowel disease in the era of biologic therapy: a position statement from the Global Interventional IBD Group. Gastrointest Endosc 2019;89:215-37.
132. Shen B. Principles, preparation, indications, precaution, and damage control of endoscopic therapy in inflammatory bowel disease. Gastrointest Endosc Clin North Am 2022;32:597-614.
133. Shen B, Kochhar G, Navaneethan U, et al. Practical guidelines on endoscopic treatment for Crohn's disease strictures: a consensus statement from the Global Interventional Inflammatory Bowel Disease Group. Lancet Gastroenterol Hepatol 2020;5:393-405.
134. Jena A, Mohindra R, Rana K, et al. Frequency, outcomes, and need for intervention in stricturing gastrointestinal tuberculosis: a systematic review and meta-analysis. BMC Gastroenterol 2023;23:46.
135. Yamamoto H, Yano T, Araki A, et al. Guidelines for endoscopic balloon dilation in treating Crohn's disease-associated small intestinal strictures (supplement to the clinical practice guidelines for enteroscopy). Dig Endosc 2022;34:1278-96.
136. Di Nardo G, Oliva S, Passariello M, et al. Intralesional steroid injection after endoscopic balloon dilation in pediatric Crohn's disease with stricture: a prospective, randomized, double-blind, controlled trial. Gastrointest Endosc 2010;72:1201-8.
137. East JE, Brooker JC, Rutter MD, et al. A pilot study of intrastricture steroid versus placebo injection after balloon dilatation of Crohn's strictures. Clin Gastroenterol Hepatol 2007;5:1065-9.
138. Ning SB, Yang H, Li B, et al. Balloon-assisted enteroscopy-based endoscopic stricturotomy for deep small bowel strictures from Crohn's disease: first cohort study of a novel approach. Dig Liver Dis 2023.
139. Wewer MD, Karstensen JG, Burisch J. Endoscopic small bowel balloon dilations in patients with Crohn's disease: a Danish nationwide cohort study, 1997-2015. Eur J Gastroenterol Hepatol 2022;34:831-7.
140. Tilmant M, Serrero M, Poullenot F, et al. Endoscopic balloon dilation of colorectal strictures complicating Crohn's disease: a multicenter study. Clin Res Hepatol Gastroenterol 2021;45:101561.
141. Lan N, Shen B. Endoscopic stricturotomy versus balloon dilation in the treatment of anastomotic strictures in Crohn's disease. Inflamm Bowel Dis 2018;24:897-907.
142. Pal P, Gala J, Rebala P, et al. Re-intervention rates and symptom-free survival at 1 year after endoscopic versus surgical management of strictures in Crohn's disease: a propensity matched analysis of a prospective inflammatory bowel disease cohort. J Gastroenterol Hepatol 2024;39:353-9.
143. Krauss E, Agaimy A, Gottfried A, et al. Long term follow up of through-the-scope balloon dilation as compared to strictureplasty and bowel resection of intestinal strictures in crohn's disease. Int J Clin Exp Pathol 2014;7:7419-31.
144. Lee KE, Lim F, Faye AS, et al. Endoscopic balloon dilation is cost-effective for crohn's disease strictures. Dig Dis Sci 2022;67:5462-71.
145. Manning RJ, Lewis C Jr. Inflammatory ileal polyps in Crohn's disease presenting as refractory iron deficiency anemia. Gastrointest Endosc 1986;32:122.
146. Hirten R, Cohen BL, Colombel JF. Colonic pseudopolyps resulting in iron deficiency anemia. Clin Gastroenterol Hepatol 2017;15:A27.
147. Romero C, Sirsi S, Asarian A, et al. Giant inflammatory polyposis, a phenomenon of inflammatory bowel disease, presenting as acute large bowel obstruction mimicking colonic neoplasm. J Surg Case Rep 2017.
148. Loras C, Andújar X, Gornals JB, et al. Self-expandable metal stents versus endoscopic balloon dilation for the treatment of strictures in Crohn's disease (ProtDilat study): an open-label, multicentre, randomised trial. Lancet Gastroenterol Hepatol 2022;7:332-41.
149. Mohapatra S, Sankaramangalam K, Lopimpisuth C, et al. Advanced endoscopic resection for colorectal dysplasia in inflammatory bowel disease: a meta-analysis. Endosc Int Open 2022;10:E593-601.
150. Ribeiro T, Mascarenhas M, Afonso J, et al. Artificial intelligence and colon capsule endoscopy: automatic detection of ulcers and erosions using a convolutional neural network. J Gastroenterol Hepatol 2022;37:2282-8.
151. Brodersen JB, Jensen MD, Leenhardt R, et al. Artificial intelligence-assisted analysis of pan-enteric capsule endoscopy in patients with suspected Crohn's disease: a study on diagnostic performance. J Crohns Colitis 2024;18:75-81.
152. Lu K, Tong Y, Yu S, et al. Building a trustworthy AI differential diagnosis application for Crohn's disease and intestinal tuberculosis. BMC Med Inform Decis Mak 2023;23:160.
153. Iacucci M, Parigi TL, Del Amor R, et al. Artificial intelligence enabled histological prediction of remission or activity and clinical outcomes in ulcerative colitis. Gastroenterology 2023;164:1180-8.
154. Iacucci M, Cannatelli R, Parigi TL, et al. PICaSSO group. A virtual chromoendoscopy artificial intelligence system to detect endoscopic and histologic activity/remission and predict clinical outcomes in ulcerative colitis. Endoscopy 2023;55:332-41.
155. Fan Y, Mu R, Xu H, et al. Novel deep learning-based computer-aided diagnosis system for predicting inflammatory activity in ulcerative colitis. Gastrointest Endosc 2023;97:335-46.
156. Gui X, Bazarova A, Del Amor R, et al. PICaSSO Histologic Remission Index (PHRI) in ulcerative colitis: development of a novel simplified histological score for monitoring mucosal healing and predicting clinical outcomes and its applicability in an artificial intelligence system. Gut 2022;71:889-98.

157. Stidham RW, Cai L, Cheng S, et al. Using computer vision to improve endoscopic disease quantification in therapeutic clinical trials of ulcerative colitis. Gastroenterology 2024;166:155-67.
158. Shen B, Renee Adorno-Garayo C. Initial safety and efficacy of a novel drug-coated balloon for treatment of benign intestinal strictures. iGIE 2024;3:10-4.
159. Vieujean S, Loly JP, Boutaffala L, et al. Mesenchymal stem cell injection in Crohn's disease strictures: a phase I-II clinical study. J Crohns Colitis 2022;16:506-10.
160. Kochhar GS, Shen B. Use of over-the-scope-clip system to treat ileocolonic transverse staple line leak in patients with Crohn's disease. Inflamm Bowel Dis 2018;24:666-7.
161. Lescaille Y, Rosh JR, Kiran RP, et al. Successful closure of the tip of the "J" fistula of the ileal pouch with double over-the-scope clips. ACG Case Rep J 2021;8:e00566.

*Abbreviations: ASGE, American Society for Gastrointestinal Endoscopy; CAN, colitis-associated neoplasia; CD, Crohn's disease; CI, confidence interval; CMV, cytomegalovirus; CRC, colorectal cancer; CTE, CT enterography; EBD, endoscopic balloon dilation; HD, high definition; IBD, inflammatory bowel disease; IUS, intestinal US; MRE, magnetic resonance enterography; PSC, primary sclerosing cholangitis; UC, ulcerative colitis; VCE, video capsule endoscopy; WL, white light.*

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