Surveillance and management of dysplasia in patients with inflammatory bowel disease

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INTRODUCTION

Because the risk for colorectal cancer (CRC) is increased in patients with inflammatory bowel disease (IBD), the goal of surveillance colonoscopy is to detect dysplasia, the precursor of colorectal cancer. We recommend surveillance for dysplasia and colorectal cancer in patients with IBD, and our approach is generally consistent with multiple societies worldwide [1-7], including updated guidance from the American Gastroenterological Association [6].

The epidemiology, risk factors, and pathology of colon cancer in IBD and the evidence supporting a role for cancer surveillance will be reviewed here. Methods for cancer surveillance will also be discussed. The definition, risk factors, clinical manifestations, diagnosis, and management of IBD are discussed separately.

- (See "Definitions, epidemiology, and risk factors for inflammatory bowel disease".)
- (See "Clinical manifestations, diagnosis, and prognosis of ulcerative colitis in adults".)
- (See "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults".)
- (See "Overview of the medical management of mild (low risk) Crohn disease in adults".)
- (See "Management of the hospitalized adult patient with severe ulcerative colitis".)
- (See "Surgical management of ulcerative colitis".)

EPIDEMIOLOGY

The risk of colorectal cancer (CRC) in patients with inflammatory bowel disease is increased compared with the general population. In a population-based study of over 96,000 patients with inflammatory bowel disease (IBD), the overall risk of CRC was 1.29 cases per 1000 person-years [8].

The mean age of developing CRC in the setting of IBD is lower than for sporadic CRC (40 to 50 years versus 60 years) [9,10]. (See "Colorectal cancer: Epidemiology, risk factors, and protective factors", section on 'Epidemiology'.)

Male sex may be a risk factor for colorectal cancer in IBD patients. In one population-based study of more than 7000 patients with IBD, males had a 60 percent higher risk of CRC (RR 1.6 95% CI 1.2-2.2) compared with females [11]. The effect of sex was seen only after ten years of follow-up and limited to patients diagnosed before age 45. This difference may be explained by variation in the extent of inflammation, or by factors related to patient behaviors that affect compliance with medication and surveillance [12].

Ulcerative colitis — The association of ulcerative colitis (UC) and colorectal cancer depends mainly upon the duration, extent, and activity of disease [8,13-18]. In a population-based cohort study including over 96,000 patients with IBD (with >10 years follow up in >50 percent of the cohort), patients with extensive colitis (defined by the Montreal classification as disease extending proximal to the splenic flexure) had an increased risk of CRC compared with individuals from the general population who were matched for age, sex, year of birth, and place of residence (521 versus 343 cases of CRC of per 1000 person-year follow up; adjusted hazard ratio [aHR] 1.88, 95% CI 1.72-2.07) [8]. In contrast, the risk of CRC in patients with ulcerative proctitis or left-sided colitis was not significantly higher compared with the general population (aHR 0.97, 95% CI 0.76-1.25 and aHR 0.90, 95% CI 0.72-1.14, respectively).

The risk factors for CRC in patients with UC are [19]:

- **Presence and severity of inflammation** – The presence and severity of inflammation appear to be important markers of risk [20-22]. In a meta-analysis of four studies including 1025 patients with ulcerative colitis, mucosal inflammation (including both histologic and endoscopic inflammation) was associated with an increased risk of colorectal neoplasia (OR 3.5, 95% CI 2.6-4.8) [22].

  In a case-control study, endoscopic features of severe inflammation, such as pseudopolyps and strictures, were associated with an increased risk of colorectal neoplasia (OR 2.29, 95% CI 1.28-4.11 and OR 4.62, 95% CI 1.03-20.8, respectively) [23]. However, data on pseudopolyps as a risk factor for dysplasia have been mixed [24,25]. A case-control study (in which cases and controls were matched for the extent and duration of UC) also found

that the risk of CRC was increased in patients with a history of inflammatory pseudopolyps [24]. In a subsequent study of 462 patients with IBD, pseudopolyps were not associated with increased risk of CRC after a median follow up of nearly five years [25]. (See 'Inflammatory pseudopolyps' below.)

- **Age at disease onset/disease duration** – Younger age at disease onset/duration of disease appears to be a risk factor in patients with extensive colitis [8,26]. In one series, the absolute risk of CRC in patients with extensive colitis was 30 percent after 35 years of disease [26]. The risk was increased in those with the onset of symptoms prior to age 15 years. However, in other reports, the age of onset of colitis did not increase the risk of CRC after adjusting for the longer period of time that young patients were at risk and the extent of the disease [27].

- **Ileitis** – One study suggested that ileitis (in which mucosal inflammation involves the terminal ileum) may be an independent risk factor for CRC [28]. However, other studies have not confirmed this association [29,30]. (See "Endoscopic diagnosis of inflammatory bowel disease in adults", section on 'Direct visualization'.)

Extensive colitis — Patients with extensive colitis, defined by the Montreal Classification as disease extending proximal to the splenic flexure, have the greatest risk of CRC. Compared to an age-matched population, the risk begins to increase 8 to 10 years following the onset of symptoms [8,31,32]. In a meta-analysis, cumulative risks of CRC after 10, 20, and greater than 20 years of disease were 1, 2, and 5 percent, respectively. High-risk groups were patients with extensive colitis and an IBD diagnosis before age 30 (SIR 6.4, 95% CI 2.4-17.5 and 7.2, 95% CI 2.9-17.8, respectively) [33]. In older epidemiologic studies, the incidence was higher than in recent decades [26,31,34-36].

Left-sided colitis — Most studies have found that the risk of CRC increases after 15 to 20 years (approximately one decade later than in extensive colitis) in patients with colitis confined to the left colon (ie, distal to the splenic flexure) [37,38].

Proctitis — Patients with ulcerative proctitis and proctosigmoiditis are probably not at increased risk for CRC [26].

Crohn disease — The risk of CRC in longstanding Crohn disease (CD) involving the colon is probably comparable to UC [27,39,40]. However, not all studies reached these conclusions and thus the magnitude of risk in patients with CD remains unsettled. In a population-based study, the relative risk of colon cancer was 2.5 in patients with CD and 5.6 in those with disease restricted to the colon [27]. The relative risk was even greater in patients who were less than 30

years of age at the time of diagnosis (RR 21, compared with those diagnosed after age 30). Similar findings have been reported in other studies [41].

CRC in CD is observed in a similar time frame as in UC [42,43]. This was illustrated in one series that included 80 patients with CRC complicating UC or CD [42]. The median duration of disease prior to the diagnosis of CRC was comparable for CD and UC (15 and 18 years, respectively). The median age at diagnosis of CRC was 55 years in CD and 43 years in UC. One series found that CD patients undergoing surgery for cancer had more advanced CRC than patients with UC [44].

Ileal pouch anal anastomosis — The incidence of CRC in patients with IBD who have undergone restorative proctocolectomy with an ileal pouch anal anastomosis (IPAA) is low [45]. In a case-control study that included 1200 patients with IBD (1053 with UC, 46 with CD, and 101 with indeterminate colitis) and IPAA, the cumulative incidence for pouch carcinoma at 5, 10, 15, and 20 years was 0.6, 1.4, 2.1, and 3.3 percent, respectively. The only risk factors for pouch neoplasia were a prior history of colorectal dysplasia and carcinoma (HR 3.8, 95% CI, 1.4-10.2 and HR 24.7 95% CI, 9.6-63.4, respectively). (See "Restorative proctocolectomy with ileal pouch-anal anastomosis: Laparoscopic approach", section on 'Patient selection criteria for laparoscopic RPC-IPAA'.)

Primary sclerosing cholangitis — An increased risk of CRC has been observed in patients with UC complicated by primary sclerosing cholangitis (PSC) [8,14]. CRC in patients with PSC was more likely to occur in the right colon, suggesting a possible role of bile acids in oncogenesis (a hypothesis supported by studies showing a protective effect of ursodeoxycholic acid) [46,47]. (See "Primary sclerosing cholangitis: Inflammatory bowel disease and colorectal cancer".)

MOLECULAR PATHOGENESIS

The pathogenesis of colon cancer in inflammatory bowel disease (IBD) differs from sporadic colorectal cancer (CRC), and distinct genetic features are present in colorectal tumors in IBD patients [48-50]. (See "Molecular genetics of colorectal cancer".)

The genetic features of IBD-associated tumors represent potential therapeutic targets and could be used to develop disease-specific diagnostic markers [48,50,51]. In genomic analyses of IBD-associated cancers, there were lower rates of *APC* and *KRAS* mutations compared with sporadic cancers while alterations in *TP53*, *IDH1*, and *MYC* were more frequent [48,50].

Loss of heterozygosity for the p53 gene and src activation occur earlier in cancers associated with IBD than in sporadic CRC [52-54]. Src activity in UC correlates with the degree of dysplasia [55].

ENDOSCOPIC AND HISTOLOGIC FINDINGS

Colorectal cancer — Colorectal cancer (CRC) complicating inflammatory bowel disease (IBD) may appear mass-like, nodular, ulcerated, or plaque-like [56]. As in sporadic colorectal cancer, most lesions in the colon are adenocarcinomas [57]. (See "Pathology and prognostic determinants of colorectal cancer".)

IBD-related cancer occurs in areas with active endoscopic and/or histologic inflammation [58].

Dysplasia — Dysplastic epithelium is one of the most important biomarkers for malignancy and provides the rationale for surveillance. Dysplasia is a precursor to IBD-associated colorectal cancer.

While dysplasia in IBD can be found at distant sites from the cancer, dysplasia in sporadic colon cancer is usually associated with a discrete polyp without inflammation. Synchronous tumors are more common in IBD than in sporadic CRC and can be found in the colon, rectum, anus, and internal or external fistulous tracts [55,57].

Endoscopic description — It is generally accepted that most dysplasia in IBD is endoscopically visible [59-61]. Terms such as dysplasia-associated lesion or mass (DALM) and adenoma-like or non-adenoma-like DALM, should be abandoned in favor of describing lesions using the Paris classification, modified by the Surveillance for Colorectal Endoscopic Neoplasia Detection and Management in Inflammatory Bowel Disease Patients International Consensus (SCENIC) group to incorporate features specific to IBD [1,4,62]. Lesion location should be identified as within or outside an area of known colitis and lesion description should include the following (table 1):

- **Morphology** – Polypoid (pedunculated or sessile) or nonpolypoid (slightly elevated, flat, or depressed).
- **Borders** – Distinct or indistinct.
- **Features of submucosal invasion (if present)** – Depressions, overlying ulceration or failure to lift with attempted submucosal injection.

Using these descriptors, lesions can be classified as endoscopically resectable or endoscopically unresectable. Endoscopically resectable lesions have the following characteristics:

- Distinct margins (when viewed with chromoendoscopy) [63].
- The lesion appears to be completely removed on visual inspection after endoscopic resection.

- Histologic examination of the resected specimen is consistent with complete removal (figure 1).

Kudo pit pattern classification has not routinely been applied to lesion characterization in colitis surveillance, since regenerative mucosa can demonstrate pit pattern III and IV without any associated dysplasia (figure 2) [64]. Identifying a Kudo pit pattern I or II may have role in ruling out neoplasia [65,66].

Histologic classification — In the United States, terminology for histologic classification of dysplasia in IBD is as follows [67]:

- Negative
- Indefinite
- Positive (with subgroups of low-grade and high-grade dysplasia)

In other parts of the world, but particularly in parts of Europe and Asia, pathologists prefer the Modified Vienna Classification [68].

Distinguishing dysplasia from reactive changes — Histologically, dysplasia may be difficult to distinguish from epithelial regeneration in the setting of mucosal inflammation or ulceration [69]. Dysplasia should be confirmed by a pathologist with expertise in IBD since interobserver variability is substantial, especially for discriminating highly reactive changes from true dysplasia. Common architectural and cytologic abnormalities seen in dysplastic epithelium include [67,70-72]:

- Increased mitoses (typical and atypical)
- Increased nuclear size
- Variation in the size and shape of nuclei (pleomorphism)
- Altered nuclear polarity
- Hyperchromaticity
- Lack of surface maturation
- Stratification of nuclei
- Abrupt transition
- Back to back gland pattern, cribriform

Regenerative changes are usually most prominent at the bases of the crypts, show evidence of surface maturation, and do not exhibit architectural disturbances [67]. One study found that immunostaining for alpha-methylacyl-Coa-racemase, a mitochondrial and peroxisomal enzyme overexpressed in many types of cancers, was highly specific for detecting dysplasia and distinguishing regenerating epithelium from true dysplasia [73].

GOAL OF SURVEILLANCE

The goal of surveillance for patients with inflammatory bowel disease (IBD) is to detect dysplasia, which is associated with a high risk of colorectal cancer (CRC) and to reduce mortality in those who develop colon cancer [42,74-76]. Despite the lack of randomized controlled trials, screening colonoscopy is recommended by multiple societies and is the standard of care [5,77-79]. In a large cohort study of IBD patients, the incidence of colon cancer was higher in those who did not have a colonoscopy within 6 to 36 months of the cancer diagnosis compared with those who did have surveillance (2.7 versus 1.6 percent). This study also demonstrated improved survival in patients with IBD undergoing colonoscopy compared to those without surveillance (OR 0.34, 95% CI 0.12-0.95) [80].

The body of literature supporting the role of colonoscopy for surveillance in IBD patients is mainly derived from case series, case-control studies, and population-based cohort studies, which suggest that surveillance results in an earlier cancer stage at diagnosis and improved CRC-related survival [80-83]. In a systematic review of four observational studies of patients with inflammatory bowel disease, the surveillance group had fewer deaths from CRC compared with no surveillance (8 versus 22 percent; OR 0.36, 95% CI 0.19-0.69) [84].

Strategies to improve detection of dysplasia are warranted to reduce the risk of interval cancer in IBD because advanced CRC can occur despite surveillance [15,85]. In one study of over 1200 patients with UC or Crohn disease enrolled in a surveillance colonoscopy program, 1.3 percent were diagnosed with CRC; 30 percent of CRC cases were determined to be interval cancers [86]. An analysis of a prospectively collected surveillance database demonstrated that over 50 percent of the cancers were interval cancers [15]. These studies were based on a variety of surveillance methods, some of which are no longer commonly used.

OUR APPROACH TO SURVEILLANCE

Patient selection and timing — In all patients with ulcerative colitis (UC) and Crohn disease (CD) involving one-third of the colon or more, we perform screening colonoscopy eight years after disease or symptom onset to initiate surveillance for colorectal neoplasia. Surveillance remains the standard of care, although reduction in mortality due to surveillance has not been clearly established. (See 'Goal of surveillance' above.)

Ideally, surveillance colonoscopy is performed when the patient has achieved clinical and endoscopic remission. Endoscopic disease activity can be documented using an endoscopic scoring system [87] (see "Endoscopic diagnosis of inflammatory bowel disease in adults"):

- For UC, the Mayo endoscopic subscore is commonly used as a target for treatment with a proposed remission score of 0 to 1 (calculator 1) [88]. The Ulcerative Colitis Endoscopic Index of Severity (UCEIS) and Ulcerative Colitis Colonoscopic Index of Severity (UCCIS) are validated endoscopic scores; UCEIS has a proposed remission score of ≤1.

- For CD, the Simple Endoscopic Score for Crohn Disease (SES-CD) has been used with a proposed remission score of ≤3 [89].

At the time of surveillance colonoscopy, we also obtain histologic staging biopsies to assess mucosal healing and the extent of disease activity.

We also recommend initial screening colonoscopy at eight years after disease onset for patients with isolated proctitis or disease involving less than one-third of the colon, to reassess disease extent as colitis may progress over time. In a study of a pathology database, the diagnosis of colorectal cancer (CRC) was delayed or missed in 17 to 35 percent of inflammatory bowel disease patients when screening was delayed until 8 to 10 or even 15 years, prompting many societies to adopt a shorter duration of disease at which to recommend starting surveillance [90,91].

Initiation of screening is recommended at the time of diagnosis in patients with a history of primary sclerosing cholangitis. Screening can be discussed with patients with a strong family history of CRC (ie, first-degree relative diagnosed before age 50) and offered depending on the age and preference of patient [1,2,92].

Patients who had proctocolectomy and ileal pouch anal anastomosis (IPAA) for the indication of dysplasia or colon cancer should undergo surveillance pouchoscopy beginning one year after surgery because of the risk of developing dysplasia of the pouch [7,93]. Pouch surveillance is continued annually for such patients. (See "Surgical management of ulcerative colitis", section on 'Pouch dysplasia/cancer' and "Management of acute and chronic pouchitis".)

For patients with ileoanal pouch but without a history of dysplasia or CRC, the approach to pouch surveillance is informed by the presence of other risk factors for dysplasia. Patients with any of the following risk factors should undergo pouch surveillance every one to three years [7]:

- History of primary sclerosing cholangitis
- Chronic pouchitis or chronic cuffitis
- CD of the pouch
- Greater than eight-year history of UC
- Family history of colon cancer in a first degree relative

For patients without a history of or risk factors for dysplasia, we perform surveillance pouchoscopy every three years [94].

Data have suggested that dysplasia or cancer of the pouch is uncommon in patients at average risk. In a systematic review of 33 studies including 8403 patients who had IPAA with variable duration of follow up, the pooled prevalence of colon cancer or dysplasia in the ileoanal pouch was 0.5 percent and 0.8 percent, respectively [94].

Surveillance for and management of anal transitional zone (ATZ) dysplasia after stapled IPAA is discussed separately. (See "Restorative proctocolectomy with ileal pouch-anal anastomosis: Laparoscopic approach", section on 'Anal transitional zone dysplasia'.)

METHODS FOR SURVEILLANCE

Chromoendoscopy — Most society guidelines advocate for high-definition endoscopy with surface chromoendoscopy as the strategy that optimizes dysplasia detection [1,2,4,95]. Although additional long-term studies are awaited, we believe that the single technique that has shown the highest yield for dysplasia detection is chromoendoscopy with targeted biopsies. Chromoendoscopy involves the topical application of indigo carmine or methylene blue to enhance mucosal irregularities and facilitate targeted biopsies. The technique and equipment needed to performing chromoendoscopy is discussed in detail separately. (See "Chromoendoscopy".)

The Surveillance for Colorectal Endoscopic Neoplasia Detection and Management in Inflammatory Bowel Disease Patients International Consensus (SCENIC) panel preferred chromoendoscopy over high-definition white light (HDWL) colonoscopy, based primarily on one observational study of 75 patients with inflammatory bowel disease (IBD) that showed higher rates of dysplasia detection with chromoendoscopy compared with HDWL colonoscopy (21 versus 9 percent) [4,96-102].

Some experts have cautioned against the widespread adoption of chromoendoscopy for surveillance before outcome studies have demonstrated its efficacy in clinical practice, its long-term benefit, or before additional data on chromoendoscopy versus high-definition white light endoscopy are available [85,103,104].

Chromoendoscopy appears to be superior to standard-definition white light colonoscopy, but its superiority over high-definition white light endoscopy is less clear:

- A 2015 meta-analysis of data derived from randomized parallel-group, prospective tandem, and retrospective two-group studies, demonstrated an incremental yield (ie, 4 to 11 percent) in the number of patients with dysplasia during colonoscopy with chromoendoscopy as compared with standard white light colonoscopy (RR 1.8, 95% CI 1.2-2.6, and absolute risk 6 percent, 95% CI 3-9) [4].

- A 2016 systematic review and meta-analysis of 10 randomized trials found an increased likelihood of detecting dysplasia with chromoendoscopy compared with other techniques (RR 1.37, 95% CI 1.04-1.79), but on subgroup analysis this effect was confirmed only for chromoendoscopy compared with standard white light endoscopy (RR 2.12, 95% CI 1.15-3.91) [105]. Pooled data from low-quality randomized trials did not show a difference between chromoendoscopy and high-definition white light endoscopy, narrow band imaging, or other advanced imaging techniques in detecting dysplasia [106]. (See 'Other techniques' below.)

- Subsequently, data from two trials suggested that chromoendoscopy was superior to HD-WLE for detecting dysplastic lesions when the procedures included both random and targeted biopsies [107,108].

Chromoendoscopy may also be more cost-effective as compared with standard-definition white light endoscopy [109]. The main barrier to implementation may be availability of the contrast dyes. (See "Chromoendoscopy", section on 'Indigo carmine'.)

In the SCENIC review of eight studies including 785 IBD patients, chromoendoscopy with targeted (± random) biopsies increased the duration of the procedure by an average of 10.7 minutes (95% CI 9.1-12.4 minutes) compared with white light colonoscopy with targeted (± random) biopsies [4]. In a trial including 305 patients with IBD, chromoendoscopy with random and targeted biopsies increased the procedure duration by an average of seven minutes compared with HD-WLE [107]. However, chromoendoscopy resulted in a higher ratio for detecting visible lesions per 10 minutes of withdrawal time (0.24 vs 0.16).

Role of random biopsies — Our practice is to take only targeted biopsies when using chromoendoscopy. At the time of surveillance colonoscopy, we may also take biopsies to assess mucosal healing or the extent of inflammation. (See 'Patient selection and timing' above.)

If a clinician is comfortable performing chromoendoscopy, random biopsies are not required. The SCENIC panelists, however, did not reach consensus on this issue: 60 percent voted to abandon random biopsy when using chromoendoscopy, and 25 percent voted to perform random biopsies given the concern for missing dysplasia in a small proportion of patients [4]. In the SCENIC analysis, dysplasia was detected with random biopsies in approximately 10 percent

of patients undergoing either chromoendoscopy or high-definition white light colonoscopy, and on targeted biopsies in the other 90 percent.

The technique of chromoendoscopy plus random biopsies likely maximizes dysplasia detection, and may be considered for a select, higher risk group [107,110,111]. A study evaluating the role of random biopsy after chromoendoscopy in 1000 patients demonstrated the following yields for dysplasia detection: 0.2 percent per-biopsy, 1.2 percent per-colonoscopy, and 12.8 percent per-patient [110]. On multivariate analysis, random biopsy-only detected dysplasia was associated with a personal history of dysplasia (OR 12.7, 95% CI 4.9-33.3), concomitant primary sclerosing cholangitis (OR 4.1, 95% CI 1.3-12.9), or a tubular appearing colon (OR 7.0, 95% CI 2.2-22.5). In a trial of 305 patients who had a total of 9760 random biopsies during chromoendoscopy or high definition-white light colonoscopy, dysplasia was detected in biopsy specimens from nine patients (yield of dysplasia: 0.092 percent), but detection rates were not significantly different between groups [107]. In a cohort study of 300 patients with dysplasia, risk factors for detecting dysplasia in random biopsies included longer disease duration (OR 1.04, 95% CI, 1.01-1.07), active inflammation (OR 2.89, 95% CI, 1.26-6.67), and history of PSC (OR 3.66, 95% CI, 1.21-11.08) [111]. These data support obtaining random biopsies during surveillance colonoscopy in high-risk groups (eg, patients with PSC).

When using a random biopsy protocol for surveillance examination, multiple random biopsies are required to adequately sample the colon. Four biopsies are obtained every 10 cm from the cecum to the rectum for a total of a minimum of 33 biopsies [112]. Additional biopsies are taken in the sigmoid colon and rectum. In addition, areas of mucosal irregularity should be biopsied. The SCENIC analysis calculated approximately one in a thousand random biopsies detects dysplasia [4].

The use of jumbo forceps has the potential to improve the dysplasia detection rate. A study comparing eight paired biopsy specimens from the rectosigmoid, obtained by either jumbo or standard large-capacity forceps, concluded that the jumbo forceps were superior for obtaining diagnostically adequate surveillance biopsy specimens (67 versus 48 percent) [113].

High definition-white light colonoscopy — While we advocate the use of chromoendoscopy, some practitioners prefer high-definition white light colonoscopy (with targeted and random biopsies) and can obtain high diagnostic yield for dysplasia with this technique. (See 'Role of random biopsies' above.)

Most society and consensus guidelines recommend high-definition white light colonoscopy with targeted and random biopsy where the yield of chromoendoscopy is decreased or the mucosa

is poorly visualized, such as with inadequate preparation, active inflammation, in the setting of pseudopolyps and strictures, or where chromoendoscopy expertise is not available [1,2,4,6,95].

In a trial of 270 patients with inactive IBD undergoing surveillance, the high definition-white light colonoscopy technique was noninferior to either dye spraying chromoendoscopy or virtual chromoendoscopy (using iSCAN technology) for detection of colonic dysplastic lesions [66].

Other techniques — Narrow band imaging (NBI) is an endoscope-based image-enhanced technology that enhances the fine structure of the mucosa without the use of dyes. However, it does not enhance dysplasia detection [4,114-116].

NBI in combination with magnification endoscopy may play a role in characterization of detected lesions [117]. (See "Magnification endoscopy".)

MANAGEMENT OF ENDOSCOPIC FINDINGS

Patients with dysplasia

Polypoid dysplasia — For endoscopically resectable dysplastic polyps that are not associated with dysplastic changes in flat mucosa elsewhere in the colon, we suggest removing the dysplastic polyp endoscopically, obtaining biopsies adjacent to the resection site when indicated, and close surveillance to ensure complete resection (algorithm 1) [4,118,119]:

- **Polyps 10 mm or greater in size** – For larger lesions or lesions removed piecemeal, surveillance colonoscopy should be performed within one to six months, as well as 12 months after the index resection, and biopsy specimens of the resection site should be obtained to document eradication of dysplastic tissue. At least annual surveillance should be performed thereafter [1,4].

- **Polyps smaller than 10 mm** – For smaller polypoid lesions resected en bloc, surveillance colonoscopy may be performed at the one-year interval [4].

This approach to polypoid dysplasia is informed by follow-up studies demonstrating that polypectomy with complete excision and continued surveillance provides adequate treatment of patients with endoscopically resectable polypoid dysplasia [4,120-122]. The Surveillance for Colorectal Endoscopic Neoplasia Detection and Management in Inflammatory Bowel Disease Patients International Consensus (SCENIC) panel pooled data from six studies found that during mean follow-up periods between 36 and 82 months, the incidence of CRC was 19 of 311 patients (6 percent, range 2 to 13 percent) [4]. In a meta-analysis of 10 studies that included 376 patients with ulcerative colitis (UC) who underwent resection of polypoid dysplasia prior to the

advent of chromoendoscopy with a combined 1704 years of follow-up, the pooled incidence of cancer and dysplasia were 5.3 (95% CI 2.7-10.1) and 65 (95% CI 54-78) per 1000 patient-years, respectively [122].

If the follow-up surveillance examination reveals that polyp resection was incomplete and dysplasia or cancer is histologically confirmed, surgical consultation is obtained for further management.

Some guidelines recommend taking biopsies from the mucosa immediately adjacent to the resection site to ensure that the lateral margins are free of dysplasia on histologic examination [1,3,4]. This practice is based on expert opinion. Visual inspection by trained endoscopists is likely sufficient, with a yield of 0 to 5 percent for unsuspected dysplasia [123,124].

We do not routinely take biopsies from the lateral resection margin unless there are concerns about the adequacy of the resection. As endoscopists performing inflammatory bowel disease (IBD) surveillance exams become familiar with optical diagnosis and treatment of IBD-associated dysplastic lesions, biopsies of the lateral margins can initially aid in their assessment of the completeness of resection.

Nonpolypoid dysplasia — Endoscopic management and surveillance of nonpolypoid, endoscopically resectable dysplasia is suggested after complete endoscopic resection, although data on long-term dysplasia and colorectal cancer (CRC) risk after endoscopic resection of nonpolypoid dysplastic lesions are not available [4]. Nonpolypoid lesions can be technically more difficult to remove, especially in the presence of fibrosis from prior or ongoing inflammation.

Patients with nonpolypoid dysplasia should be managed by an endoscopist with expertise in advanced endoscopic resection techniques. As with polyploid dysplasia, we perform surveillance colonoscopy within one to six months and repeat the exam in 12 months after the index resection. We obtain biopsy specimens of the resection site to document eradication of dysplastic tissue. Annual surveillance colonoscopy should be performed thereafter [1,4].

Invisible dysplasia — Invisible dysplasia is dysplasia detected on random (ie, non-targeted) biopsies of colonic mucosa without an associated visible lesion. Patients found to have invisible dysplasia on random biopsy should be referred to an IBD center that offers high definition chromoendoscopy. A discussion of risks and benefits of management strategies should be discussed with the patient.

- **Unifocal, low-grade dysplasia** – Although the management of invisible, low-grade dysplasia (LGD) remains controversial, we agree with most societies that the diagnosis of

invisible dysplasia should be confirmed by a second pathologist with expertise in interpretation of biopsies in patients with IBD and a repeat colonoscopy with high-definition chromoendoscopy should be performed by an experienced endoscopist. In addition to targeted biopsies, we also obtain additional random biopsies at the follow-up colonoscopy to maximize the yield of dysplasia.

The rates of progression from LGD to high-grade dysplasia (HGD) and cancer range from 0 to greater than 50 percent [125,126]. Studies performed in the era of chromoendoscopy demonstrate that the majority of patients with LGD will not progress to higher grades of dysplasia during three to four years of follow-up [127-130]. A meta-analysis of studies using white light colonoscopy for surveillance demonstrated a positive predictive value of flat (invisible) low-grade dysplasia of 22 percent for concurrent CRC and 36 percent for concurrent HGD and/or CRC [131].

- **Multifocal, low-grade dysplasia** – For patients with multifocal, invisible, LGD that is detected on random biopsy during surveillance colonoscopy and confirmed by a second pathologist, we recommend colonoscopy with chromoendoscopy by an experienced endoscopist [1,2,4]. Chromoendoscopy in patients with invisible dysplasia may identify a visible lesion that may be amenable to endoscopic removal. Variable rates of progression from LGD (identified by random biopsies using standard-definition colonoscopies) to high-grade dysplasia or CRC were noted in these studies:

  - 50 percent (9 of 18) progressed to a more advanced lesion (eg, HGD or CRC) at a median of 32 months [132].
  - 15 percent (7 of 46) progressed to CRC at five years [133].
  - 10 percent (3 of 29) progressed to HGD or CRC at 10 years [134].

- **High-grade dysplasia** – Patients with invisible high-grade dysplasia confirmed by a second pathologist should be managed by an endoscopist with expertise in IBD surveillance with high-definition chromoendoscopy. This approach was also advocated by the SCENIC panel, although they did not endorse either endoscopic surveillance or colectomy for these patients, as much of the literature predates the video-endoscopic era [4].

  An endoscopically resectable lesion may be managed with intensive surveillance [4], as studies demonstrate that curative resection of circumscribed lateral spreading lesions with HGD can be achieved [61,135]. For most patients, the first surveillance colonoscopy is performed in three to six months after the index examination, and then annually

thereafter. However, this can vary depending on findings at endoscopy and the judgment of the expert endoscopist.

We agree with the SCENIC consensus statement that if dysplasia is not detected on the follow-up colonoscopy, a decision regarding surveillance versus colectomy should be individualized after a discussion of risks and benefits of the different management strategies [4]. Alternatively, the European Crohn's and Colitis Organization and The American Society for Gastrointestinal Endoscopy state that HGD without an associated endoscopically visible lesion is an indication for surgery [1,2].

Inflammatory pseudopolyps — Inflammatory pseudopolyps are irregularly shaped islands of residual intact colonic mucosa that are the result of the mucosal ulceration and regeneration that occurs in IBD (picture 1 and picture 2). (See "Overview of colon polyps", section on 'Inflammatory pseudopolyps'.)

When typical features are present, inflammatory pseudopolyps do not require excision unless they cause symptoms (eg, bleeding, obstruction). While not dysplastic, they are a marker of prior severe inflammation, which is a risk factor for colon cancer in UC [24,29]. However, their presence can also complicate the recognition of dysplastic lesions. Inflammatory pseudopolyps can be recognized by their histologic features; thus, a biopsy can help make the distinction in unclear cases.

Strictures — Patients with IBD with a colorectal stricture that cannot be passed or adequately biopsied should be referred for surgical consultation for consideration of resection [1]. Strictures can complicate both colonic Crohn disease (CD) and UC, and their presence requires close surveillance due to an increased risk of CRC that has been reported in most but not all studies [23,136-138]. In one retrospective study of 293 IBD patients with colorectal strictures requiring surgery, dysplasia or cancer was found in 3.5 percent of strictures [139]. In a population-based study of 640 CD patients, the risk of developing colon cancer was higher in patients with colonic stenosis compared with those without stenosis (HR 18.8 95% CI 3.45-102.7). The probability of developing CRC for these patients was 5.5 and 7.5 percent after 5 and 10 years, respectively [140].

CHEMOPREVENTION

Although several agents have been evaluated for prevention of CRC in patients with inflammatory bowel disease (IBD), none have conclusively been shown to decrease the risk of CRC. In IBD, cancer risk is thought to be related to chronic inflammation. A drug that reduces

inflammation may lead to a reduction in colitis-associated neoplasia. Data are mixed and recommendations are based upon mainly observational studies [141].

Several drugs have been studied in the non-IBD population (particularly nonsteroidal antiinflammatory drugs and calcium) and some have also been evaluated in IBD and in the aggregate have not been found to be effective. (See "Colorectal cancer: Epidemiology, risk factors, and protective factors" and "NSAIDs (including aspirin): Role in prevention of colorectal cancer".)

- 5-aminosalicylates – We use mesalamine for chemoprevention, particularly if 5-aminosalicylates have played a role in inducing clinical remission. The European Crohn's and Colitis Organization states that 5-aminosalicylates (5-ASAs) may reduce the incidence of colorectal cancer in ulcerative colitis (UC), and is suggested for all UC patients [79]. While the data for the chemopreventive effect of 5-ASAs are conflicting, 5-ASAs are generally considered to be low risk with a good safety profile, with both an anti-inflammatory effect and potential molecular anticarcinogenic effect [13,141]. (See "Medical management of low-risk adult patients with mild to moderate ulcerative colitis", section on 'Induction of remission'.)

- Ursodeoxycholic acid – (See "Primary sclerosing cholangitis: Inflammatory bowel disease and colorectal cancer".)

- Folic acid – (See "Primary sclerosing cholangitis: Inflammatory bowel disease and colorectal cancer".)

- Other agents – While some studies have suggested that thiopurine use may decrease the risk of CRC, there are insufficient data to recommend thiopurines for chemoprevention in patients with IBD [2,17,79,142-144]. Data supporting anti-tumor necrosis factor as chemoprevention is lacking [13].

  A retrospective cohort study suggested that statin use was associated with lower risk of CRC in patients with IBD, but prospective studies are need to confirm these findings [145].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Ulcerative colitis in adults" and "Society guideline links: Crohn disease in adults" and "Society guideline links: Colorectal cancer".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Colon and rectal cancer screening (The Basics)" and "Patient education: Ulcerative colitis in adults (The Basics)" and "Patient education: Crohn disease in adults (The Basics)" and "Patient education: Colonoscopy (The Basics)")

- Beyond the Basics topics (see "Patient education: Screening for colorectal cancer (Beyond the Basics)" and "Patient education: Ulcerative colitis (Beyond the Basics)" and "Patient education: Crohn disease (Beyond the Basics)" and "Patient education: Colonoscopy (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- The risk of colorectal cancer (CRC) in patients with inflammatory bowel disease is related to the type, severity, duration, and anatomic extent of the disease. Patients with extensive colitis, defined as disease extending proximal to the splenic flexure, have the greatest risk of CRC. (See 'Epidemiology' above.)

- For surveillance, we perform chromoendoscopy with targeted biopsies because this technique has shown the highest yield for dysplasia detection. Chromoendoscopy involves the topical application of indigo carmine or methylene blue to enhance mucosal irregularities and facilitate targeted biopsies. (See 'Chromoendoscopy' above.)

- Surveillance with high-definition white light colonoscopy with targeted and random biopsies is an acceptable alternative, and multiple biopsies are required to adequately

sample the colon. We obtain four biopsies every 10 cm from the cecum to the rectum. Additional biopsies should be taken in the sigmoid colon and rectum. Alternatively, six biopsies from the right colon, transverse colon, descending colon, sigmoid, proximal, and distal rectum can be taken in patients with ulcerative colitis (UC). In addition, areas of mucosal irregularity should be biopsied. (See 'High definition-white light colonoscopy' above.)

- For most patients with left-sided or extensive UC, or Crohn colitis involving more than one-third of the colon, we perform colonoscopy at eight years after disease onset to initiate surveillance for dysplasia, and we continue surveillance examinations every one to three years. (See 'Patient selection and timing' above.)

  For patients who have undergone a subtotal colectomy with an ileostomy and have a rectum left in place (ie, a Hartmann's pouch), surveillance examination of the remaining rectum is performed every one to three years.

  For patients with an ileal pouch anal anastomosis (IPAA), surveillance pouchoscopy is performed at time intervals that are guided by the patient's risk for dysplasia:

  - For patients with a history of CRC or dysplasia, we perform pouchoscopy yearly.
  - For patients without a history of CRC or dysplasia but with other risk factors (eg, chronic pouchitis, primary sclerosing cholangitis), we perform pouchoscopy every one to three years.
  - For patients without risk factors, we perform pouchoscopy every three years.

- For endoscopically resectable dysplastic lesions that are not associated with dysplastic changes in flat mucosa elsewhere in the colon, we remove the dysplastic polyp endoscopically and follow up with close surveillance. If the follow-up examination reveals that polyp resection was incomplete and dysplasia or cancer is histologically confirmed, surgical consultation is obtained for further management. (See 'Polypoid dysplasia' above.)

- Patients found to have invisible dysplasia on random biopsy only should be referred for high definition chromoendoscopy at an expert inflammatory bowel disease center. (See 'Invisible dysplasia' above.)

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Topic 4079 Version 36.0

GRAPHICS

Terminology for reporting findings on colonoscopic surveillance of patients with inflammatory bowel disease

Term	Definition
Visible dysplasia	Dysplasia identified on targeted biopsies from a lesion visualized at colonoscopy
Polypoid	Lesion protruding from the mucosa into the lumen ≥2.5 mm
▪ Pedunculated	Lesion attached to the mucosa by a stalk
▪ Sessile	Lesion not attached to the mucosa by a stalk (entire base is contiguous with the mucosa)
Nonpolypoid	Lesion with little (<2.5 mm) or no protrusion above the mucosa
▪ Superficial elevated	Lesion with protrusion but <2.5 mm above the lumen (less than the height of the closed cup of a biopsy forceps)
▪ Flat	Lesion without protrusion above the mucosa
▪ Depressed	Lesion with at least a portion depressed below the level of the mucosa
General descriptors	
▪ Ulcerated	Ulceration (fibrinous-appearing base with depth) within the lesion
▪ Border	
• Distinct border	Lesion's border is discrete and can be distinguished from surrounding mucosa
• Indistinct border	Lesion's border is not discrete and cannot be distinguished from surrounding mucosa
Invisible dysplasia	Dysplasia identified on random (non-targeted) biopsies of colon mucosa without a visible lesion

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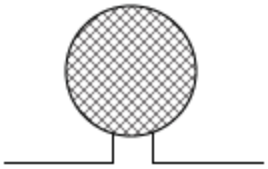
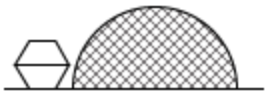
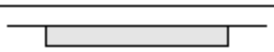
Graphic 113706 Version 1.0

Description of endoscopically visible lesions

Endoscopic appearance	Description*	Definition	Paris classification¶
Polypoid (lesion protruding from mucosa into the lumen ≥2.5 mmΔ)			
	Pedunculated	Lesion attached to mucosa by a stalk	Ip
	Sessile	Lesion not attached to mucosa by a stalk: Entire base is contiguous with mucosa	Is
Non-polypoid (lesion with little [<2.5 mm] or no protrusion above mucosa)			
	Slightly elevated	Lesion with protrusion but <2.5 mm above mucosa	IIa
	Flat	Lesion without protrusion above mucosa	IIb
	Depressed	Lesion with at least a portion depressed below the level of mucosa	IIc

Description of endoscopically visible lesions (Paris classification).[1,2]

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* Also include location (within or outside an area of known colitis), borders (distinct or indistinct), and presence of ulceration and/or other features of submucosal invasion.

¶ Morphological combinations of lesions can occur.

Δ 2.5 mm = size of closed cup of biopsy forceps.

References:

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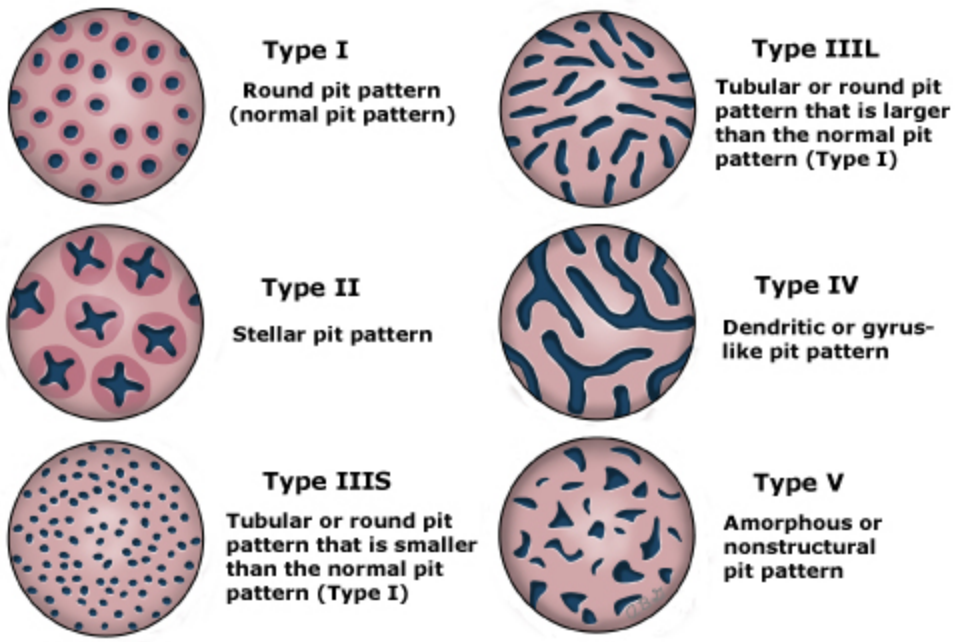
Graphic 114734 Version 1.0

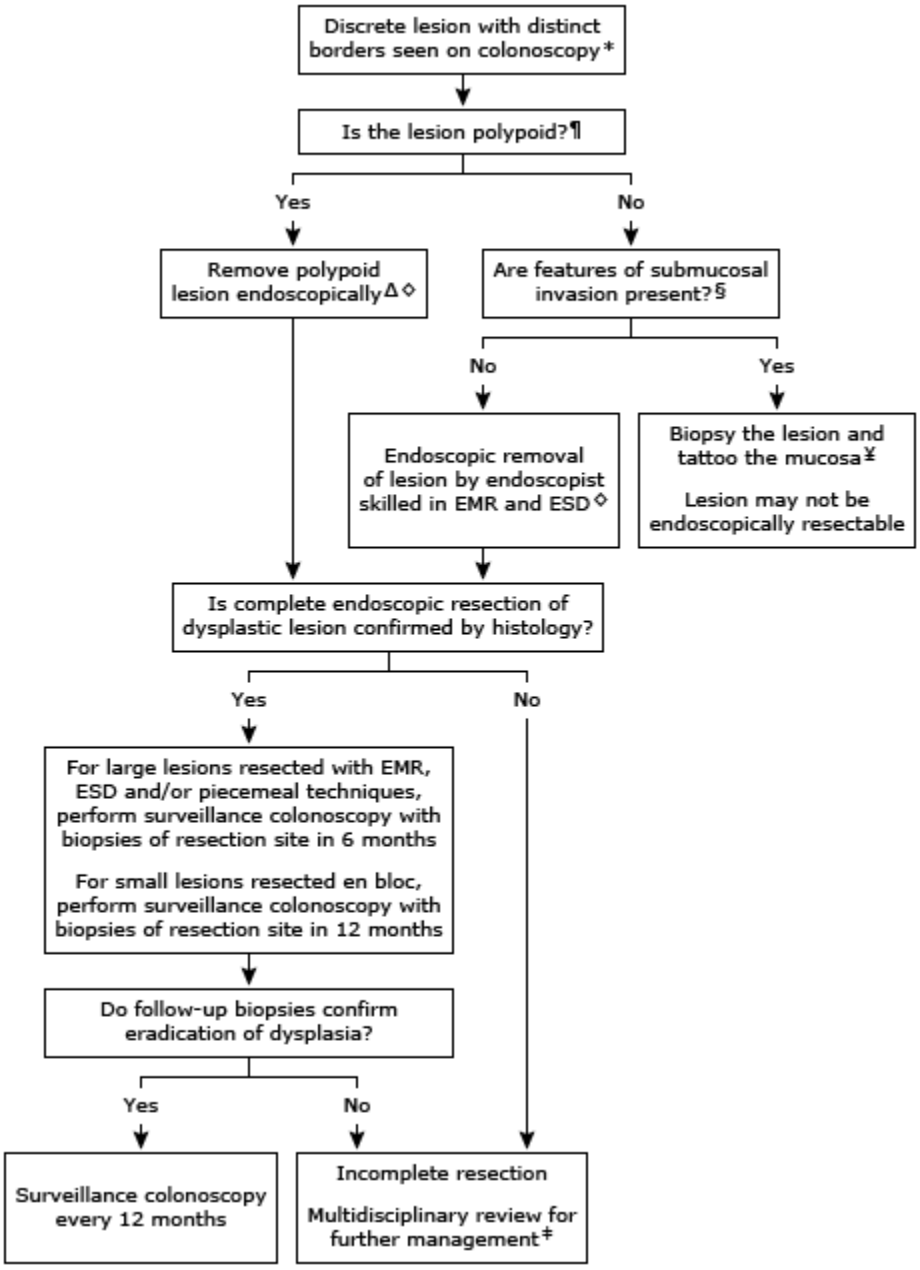
Kudo Pit Pattern Classification of colonic mucosal lesions



Pit pattern classification for colonic mucosal lesions.

Graphic 69425 Version 6.0

Management of an endoscopically visible lesion found during surveillance colonoscopy for inflammatory bowel disease



Refer to related UpToDate content.

EMR: endoscopic mucosal resection; ESD: endoscopic submucosal dissection.

* A concentrated dye (ie, indigo carmine or methylene blue) can be used to enhance visualization of the lesion.

¶ A polypoid lesion can be either sessile or pedunculated. A nonpolyoid lesion can be slightly elevated, flat, or depressed. Refer to UpToDate

content on description and classification of endoscopically visible lesions.

Δ Methods of resection include snare polypectomy or endoscopic mucosal resection (may require referral to endoscopist skilled in advanced endoscopic techniques).

◇ Following polypectomy, biopsies are taken of flat, normal appearing mucosa surrounding the resection site. Biopsies are placed in a container separate from polypectomy specimen. For larger lesions resected with EMR, ESD, and/or piecemeal techniques and that require closer surveillance, place tattoo 3 to 5 cm distal to resection site if needed.

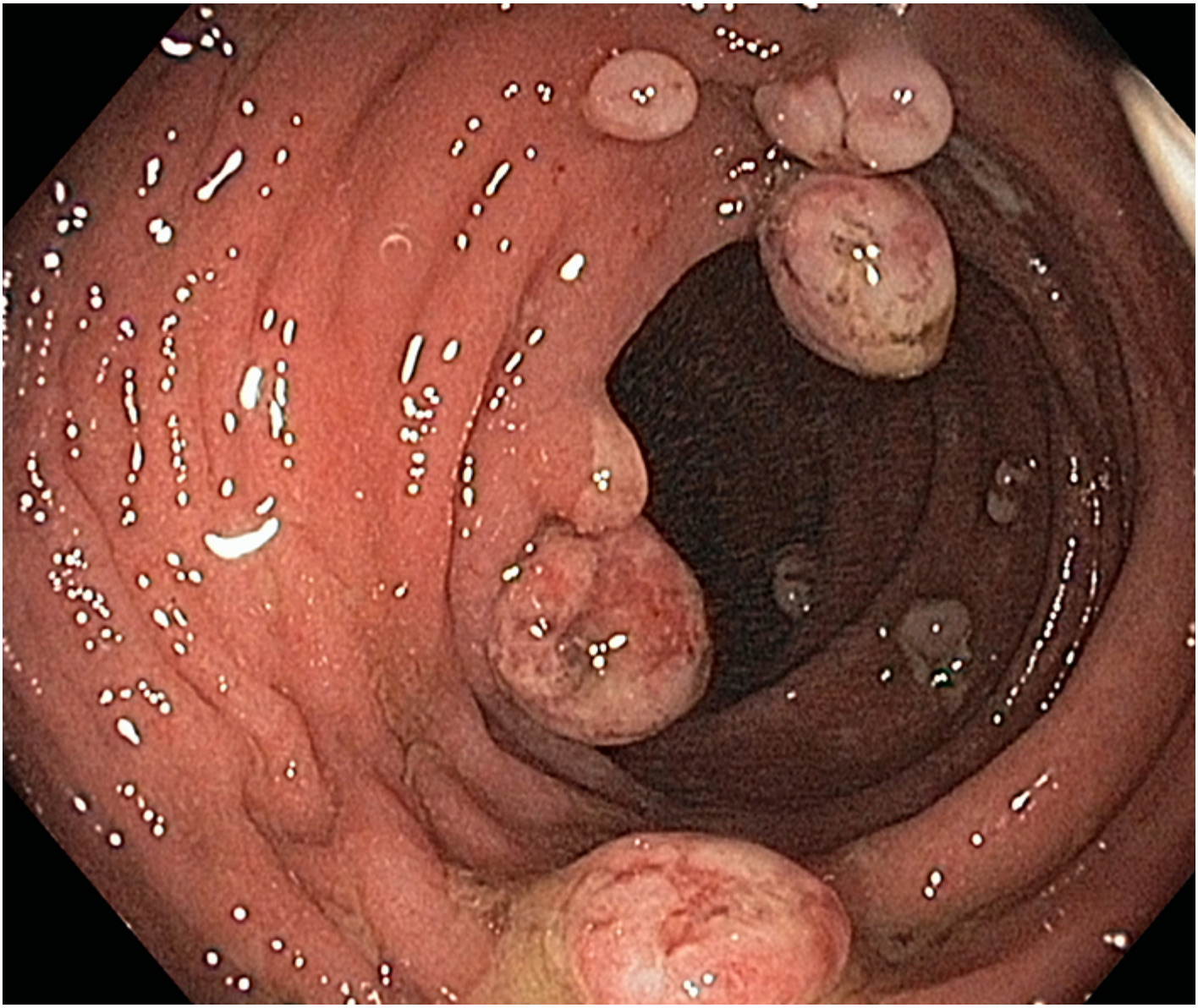
§ Features of submucosal invasion include: Depressions, failure to lift with submucosal injection, or overlying ulceration. Of note, lesions in an area of inflammation may be fibrotic and result in a falsely positive non-lifting sign. Areas of inflammation may also be ulcerated.

¥ If lesion has features of submucosal invasion, biopsy the lesion and tattoo the mucosa 3 to 5 cm distal to the site. If the lesion is discrete, refer the patient to advanced endoscopist to assess for endoscopic resection.

‡ Multidisciplinary review includes referral to advanced endoscopist and/or colorectal surgery for further management options.

Graphic 116543 Version 2.0

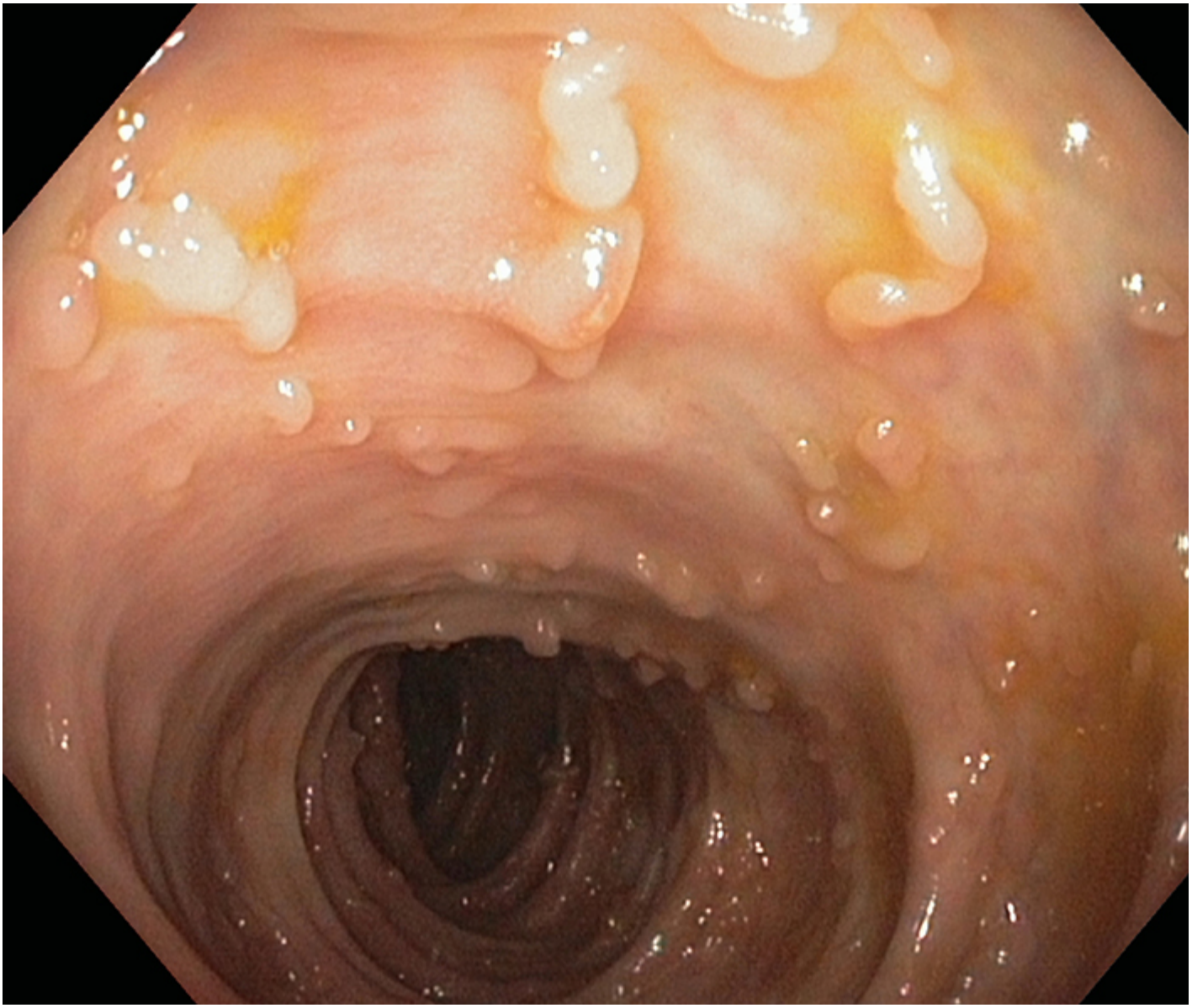
Endoscopic view of colon pseudopolyps in inflammatory bowel disease



This endoscopic image from a patient with ulcerative colitis demonstrates multiple pseudopolyps. Pseudopo may also be seen in patients with Crohn colitis.

Graphic 114458 Version 1.0

Pseudopolyps in IBD



Endoscopic view of the colon with multiple pseudopolyps

IBD: Inflammatory bowel disease

Graphic 111378 Version 1.0

Contributor Disclosures

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